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GUEST EDITORIAL

The Arteriosclerotic Abdominal Aortic Aneurysm

UNTIL recently, abdominal aortic aneurysms were considered a rarity[1], usually due to syphilis[2], characterized by a poor prognosis without therapy, and in many instances the prognosis worsened with surgical intervention.

In the year of 1953, some of the above thoughts have changed for several reasons. Firstly, effective antiluetic therapy combined with enlightened public health measures have reduced markedly the number of patients suffering from syphilitic aneurysms of the abdominal aorta. Secondly, the increasing number of the population in the sixth to eighth decades and possibly an increasing incidence of arteriosclerosis associated with a more advanced degree of the disease than observed earlier in this century have led to an actual increase in the number of patients developing arteriosclerotic aneurysms of the abdominal aorta. Thirdly, and probably the most important of all, is the fact that following the development of a therapeutic method effective in controlling a disease, afflicted patients begin to turn up at every corner. Although it would be unduly optimistic to imply that safe curative techniques are now available for the control of arteriosclerotic aneurysm of the abdominal aorta, there are at present several surgical approaches which are either genuinely palliative or actually curative. The major problem at present is that the techniques with low risk are only palliative, and the curative methods are associated with mortality rates not entirely insignificant.

The arteriosclerotic process leads to an elongation, dilatation and tortuosity of the abdominal aorta. The most advanced intimal changes are seen in its terminal 10 to 12 centimeters at times with ulceration and calcification of the intima. When aneurysmal dilatation has occurred, there is angulation of the aorta with protrusion anteriorly and to the left and sometimes an associated buckling of the left common iliac artery. Rarely does pressure develop against the vertebral bodies. The aneurysm is usually fusiform in contour but occasionally well developed saccular aneurysms are encountered. The most frequent and usually first symptom (and sign) is a progessively evident abdominal pulsation noted by the patient and by his physician. The second most common symptom is rupture usually followed by death in hours or, at the most, days. Low back pain and mild abdominal pain are occasionally noted.

The most important question raised by the patient is how long will be the period between discovery of the aneurysm and its rupture? One recorded case lived 18 years. I have observed rather helplessly a patient die within three weeks of the initial discovery of the aneurysm. However, a fair estimate is that the patient lives in reasonable comfort and fairly good health for 12-18 months following the original diagnosis. Tragically, no finding on physical examination nor any laboratory tool will delineate between the aneurysm which will hold for several years and the one which will burst in a few days.

Since the disease has such a poor ultimate prognosis and since there is little effective therapy after the onset of rupture, surgical intervention following recognition is usually the path of choice. At present there are a variety of techniques available, the most important one being resection of the aneurysm followed by restoration of arotic-iliac

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continuity by means of homologous aortic graft. The development of satisfactory methods of storage of arterial segments and evidence of their relative safety when used in man has made possible a frontal attack on the abdominal aortic aneurysm. Murray, Freeman, Bahnson, Cooley and DeBakey have, along with others in Canada and the United States, contributed to this curative approach. The operative risk is not small and the ultimate fate of the grafts unknown. Nevertheless, the hospital mortality is far less than that of the disease's natural course. Gross, who pioneered homologous aortic grafts for coarctation of the aorta, has found them to stand up satisfactorily for at least five years in replacing segments of thoracic aorta.

A second operative method of less magnitude but still of some therapeutic value is the application of a sclerosing agent to the adventitial layers of the aneurysm. Polythene film impregnated with sodium dicetyl phosphate has controlled the progress of many abdominal arteriosclerotic aneurysms but has not, to my knowledge, cured a single one. The use of a suspension of sodium dicetyl phosphate has recently been studied[3] and a number of favorable early results noted, such as relief of pain and cessation of its growth. Again, probably in no case will the fibrotic encasement eradicate the aneurysm.

There are other methods of controlling the arteriosclerotic aneurysm of the abdominal aorta such as 1) ligature of the aorta proximal to the aneurysm and distal to the renal arteries—rarely successful either for life or survival of limbs; 2) packing with wire sufficient to cause an increase in the intrasaccular thrombus but not to cause sudden complete thrombosis—a method with some advocates but with many failures and complications in the hands of other surgeons; 3) proximal aortic partial occlusion with slingshot rubber band favored by Blakemore—tricky because of an extremely narrow margin of safety between too little and too much occlusion.

In summary, the arteriosclerotic aneurysm of the abdominal aorta is being encountered relatively more frequently, is a highly lethal disease usually within two years of discovery, best treated at present by either the most radical method of resection with graft or by the most conservative method of application of a sclerosing agent to its wall. The therapeutic approaches available between the two extremes appear to have less favorable features for general application.

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THE DIAGNOSIS AND TREATMENT OF POST-TRAUMATIC CEREBROSPINAL FLUID RHINORRHOEA— A REPORT OF FOUR CASES*

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Introduction:

This report presents descriptions of four patients with this lesion whom we have seen and treated at the Medical College of Virginia Hospitals in the last five years. Three were white patients, one was colored. All four cases were adult males and they all had *post-traumatic* cerebrospinal fluid rhinorrhoea; two of the four cases developed this lesion after head injury or operative intervention on the nasal sinuses; the remaining two were caused by gunshot wounds of the head, the bullet traversing or penetrating the anterior fossa on one or both sides in each case; one of these two latter patients also had a post-traumatic pneumocephalus in which the ventricles were clearly outlined on the skull films, as in a ventriculogram. These four cases will be briefly described together with comment on the definition of the lesion, the symptoms, signs, diagnosis and complications of this always dangerous condition, and the surgical treatment of choice employed by us for its permanent correction.

Definition:

Post-traumatic cerebrospinal fluid rhinorrhoea is defined as a leak of cerebrospinal fluid from one or both sides of the nose following head trauma (or operative interference upon the paranasal sinuses) and is thus distinguished from the *spontaneous* variety occurring, it is now thought, from congenital defects in the cribriform plate and dura through which the arachnoid herniates and ruptures, often during coughing, sneezing or straining, without other trauma of any kind. Three such patients with *spontaneous* leaks (rhinorrhoea) were successfully operated upon by the late Dr. C. C. Coleman in an eighteen-month period (1943-44) at the Medical College of Virginia Hospital with elimination, permanently, of the leak in every case by implanting muscle *intrddurally* after first gently avulsing the olfactory nerve and bulb on the side of the rhinorrhoea, through a unilateral transfrontal craniotomy. These cases were reported by Drs. Coleman and Troland[1] in 1947. Oddly enough, all three of those patients were middle-aged white females, ranging from 40 to 50 years of age. CSF rhinorrhoea has also been reported, rarely, as due to a post-traumatic dural defect in the *middle* or *posterior fossa,* the fluid reaching the nose by way of the Eustachian tube. Four such cases of rhinorrhoea were reported by Ecker[2], one of which followed removal of a cystic cerebello-pontine angle tumor. Ecker emphasizes that, to avoid misdirected exploration of the *anterior* fossa of the skull, it is important to recognize these rare cases in which CSF issues from the nose and in which the dural defect lies in the *middle* or *posterior,* and not the anterior, fossa. There are a number of reports in the literature in which cerebrospinal fluid rhinorrhoea occurred incident to a large pituitary tumor which had eroded the floor of the sella (roof of the sphenoid sinus). Shea[3], and Som and Kramer[4] have reported such cases ending fatally due to meningitis. The senior author of this paper, several years ago, operated on a middle-aged white woman with a CSF rhinorrhoea incident to a chromophobe pituitary tumor. After evacuation of the partly cystic pituitary lesion, the floor of the sella turcica was firmly packed with muscle which was allowed to remain *in situ.* The rhinorrhoea promptly stopped permanently. Occasionally, nasal "polyps" are removed, following which CSF rhinorrhoea develops, suggesting that the "polyps" were probably intransal meningoceles.

SIGNS, SYMPTOMS, DIAGNOSIS AND COMPLICATIONS OF CSF RHINORRHOEA

A leak of clear fluid from the nose does not always mean a cerebrospinal fluid fistula. The "fluid" may

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be merely nasal mucus but the application of Gurdjian's handkerchief test usually solves the problem. He states that mucus from the nose when drying on a handkerchief stiffens the fabric, whereas CSF does not do so. Often the leak is not voluminous and may be easily overlooked by the patient, the nurse, or the physician. Another method of demonstrating its presence in doubtful cases is to have the patient lean forward in a sitting position, during which the jugular veins are both compressed simultaneously by the examiner; this manoeuver usually sharply accelerates the rate of flow of CSF from the nose. Indigo-carmine in the lumbar subarachnoid space can be seen coming from one or both nostrils 30 to 45 minutes later by the rhinologist, although even ordinary spinal puncture in these post-traumatic cases is somewhat risky from the standpoint of meningitis that might follow such a procedure. The fluid from the nose may be collected in a test tube and analyzed for cell count, protein and sugar content, thus proving that it is spinal fluid and not mucus. Also, inspection of the upper nares by the rhinologist usually yields the desired localizing information in the spontaneous type of case. X-ray examination of the skull in the post-traumatic cases of CSF rhinorrhoea usually shows a fracture line or multiple fracture lines in the area of the frontal sinuses, ethmoids, cribriform plate or the sphenoid sinus. Often the so-called Water's P-A X-ray view of the frontal-ethmoid-zygomatic area yields the most information. Most post-traumatic CSF rhinorrhoeas—and the lesion is seen not rarely in any busy neurosurgical clinic dealing with considerable numbers of head trauma cases of all types—heal spontaneously in a few days and nothing further is required; such is almost invariably the case with CSF *otorrhoea*. It is only the *persistent* types, that continue to drain spinal fluid from the nose after two weeks or more following trauma, that require operative repair of the leak. A rather severe headache often accompanies the leak of fluid, probably for the same reason that the occasional spinal puncture is followed by post-puncture headache. Gurdjian and Webster[5], in commenting on cerebrospinal fluid rhinorrhoea, state that the post-traumatic form may be seen during the acute stage of the head injury, or as a late complication. They believe that if the fluid is found by the rhinologist to be coming from the roof of the nose, the cranio-nasal fistula is probably by way of the cribriform plate; if the fluid seems to escape from below the middle turbinate, the fistula is probably through the frontal sinus. In one of the cases reported by Cairns[6] in 1937, the leakage of CSF through the nose took place on the side *opposite* that of the fracture in the anterior fossa of the skull. Therefore, on the basis of the above variations, it is evident that each case must be diagnosed on its own merits as an individual diagnostic problem.

Occasionally, the CSF rhinorrhoea is accompanied by intracranial aerocele. It is of interst that the first case of this type demonstrated roentgenologically was presented as a pathological curiosity by Luckett[7] and was reported in 1913: clearly demonstrated was the ventricular system in the skull films made in a patient with a basilar head injury; this publication antedated by five years the report of the deliberate introduction of air into the cerebral ventricles by Dandy, thus demonstrating—even to-day—the best way to localize operable brain tumors. The presence of air in the cranial cavity is, of itself, no basis for great concern (according to Gurdjian and Webster) except that following *trauma,* it is due to the existence of a communication between the intracranial subarachnoid or (and) ventricular spaces and the outside air. Considered in this light, it is a dangerous sign, because of the always-present risk of meningitis in such a situation. The air may be in the ventricles (as in our case #4), in the subdural space, or even in the intercerebral hemispheric space alongside the falx, reported in a case of CSF otorrhoea. Tarlov and Mule[8] published (1946) a report of post-traumatic pneumocephalus associated with cerebrospinal fluid *otorrhoea* which terminated fatally. Post-traumatic pneumocephalus without CSF leak through the nose or ear usually is absorbed spontaneously, as can be shown by a series of skull films taken every few days after the pneumocephalus is first discovered. There is, of course, always a danger of meningitis developing in these cases of CSF rhinorrhoea with or without pneumocephalus, much more so than with *otorrhoea,* in our experience. The sudden development of high fever, increase of headache, together with the finding of a spastic neck and stupor usually lead to the inevitable demonstration of cloudy spinal fluid by lumbar puncture, a development which, before the days of sulfa drugs and antibiotics, almost always heralded the rapidly-developing lethal exodus of the patient. Occasionally, the pneumocephalus will not be seen on the *initial films* made at the time of trauma, but will be demonstrated for

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the first time a week or more later after the patient has coughed or strained considerably. Such an example is our case #4 (*infra*), due to a bullet wound of the anterior fossa and right cerebral hemisphere, chiefly. It was only in the later films—12 days after trauma—that the ventricles were first found filled with air and obviously a post-traumatic pneumocephalus had developed. This patient was the most difficult one of the four cases, herein reported, to treat as he was first seen by us rather late after trauma when a certain amount of basilar infection and subarachnoid hemorrhage had been present for 10 days, associated with CSF rhinorrhoea, a spastic neck, temperature elevation, and a late development of post-traumatic ventricular pneumocephalus. Certainly the early and aggressive use of sulfa drugs, and the antibiotics as well, was fully as important in salvaging this patient as the surgery that was performed ultimately. Loyal Davis[9] states, in commenting on pneumocephalus, that sneezing, coughing, straining, or swallowing is necessary to force air through the bony, dural and arachnoidal defect into the cranial chamber. Air may fill a frontal or parietal lobe of the brain without involving the ventricles at all; it may be confined, on the other hand, to the *subarachnoid* spaces. Davis states that the cavity of air in the frontal or other lobes always communicates with the frontal or ethmoid sinus by an opening which may be so minute as to be easily overlooked. The presence of air in the intracranial chamber somewhat complicates the treatment of the CSF rhinorrhoea, but the air is usually permanently obliterated at the same time as the rhinorrhoea by the operative procedure about to be described.

Surgical and Other Treatment

The essential factor and principle to be followed in the operative repair of cases of post-traumatic CSF rhinorrhoea is the same as in the *spontaneous* type, i.e. firm application of muscle intradurally to the opening or fistula between the subarachnoid space and the bony defect in the basi-frontal region or cribriform plate. In previous years, a wide variety of procedures—operative and non-operative—were reported in the literature, the great number of different methods testifying to the fact that none of them were consistently as effective as the modern intradural implantation of muscle in the fistula, rather than gel-foam which is too absorbable. The only difference in repairing the post-traumatic and the spontaneous CSF rhinorrhoea case is that in the latter it is necessary to avulse gently the olfactory nerve and bulb from its groove on the side of the leak and then to implant muscle firmly in its place. For many years, as suggested by Adson and Uihlein[10] at the Mayo Clinic, bilateral frontal *extradural* operations were carried out in rhinorrhoea cases with sacrifice of both olfactory nerves and often ligation of the superior longitudinal sinus. All *extradural* procedures in this area are open to the criticism that the dura over the cribriform plate is very thin, adherent and friable and extremely liable to be torn when stripped from the bone extradurally. All four post-traumatic cases herein reported were permanently relieved of their CSF rhinorrhoea (as were the three cases of *spontaneous* CSF rhinorrhoea in our clinic referred to above and previously reported)—one with associated ventricular pneumocephalus—by a simple *intradural* unilateral occlusion of the post-traumatic fistulae with muscle, via transfrontal craniotomy.

A successful outcome in the treatment of CSF rhinorrhoea associated with pneumocephalus has recently been reported by Pfeil and Schear[11]. A posterior nasal pack was inserted for ten days and this alone successfully stopped the leak of fluid from the nose and the pneumocephalus, involving the ventricles and the subdural and subarachnoid spaces as well, disappeared. Sixteen months later, the patient was in good health. In another patient, an unexplained disappearance of the leak for many months followed the introduction of 2 cc. of dye (indigocarmine) in the lumbar subarachnoid space, used as a diagnostic test, but the leak again appeared spontaneously twenty months later (Kahn[12], 1951). Silver nitrate applied to the fistula intranasally has been tried with some success in other clinics, but this method has now been largely abandoned. If the CSF leak does not stop spontaneously in approximately two weeks after trauma by placing the patient in the semi-Fowler position, restriction of fluids, the constant daily administration of sulfadiazine, terramycin and penicillin, until six or seven days after the leak stops, operative intervention is usually indicated. Likewise, in those patients with associated pneumocephalus, if subsequent skull films show an *increase* in the amount of air together with a continuation of the CSF leak, operation is urgently indicated and the procedure of choice is—at present —*intradural* occlusion of the fistula—almost always

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in the basi-frontal-ethmoid-cribriform area—with muscle firmly packed in the bony defect. This method is followed more consistently by success in permanently closing the fistula and obliteration of the CSF leak and pneumocephalus (if present), than any other procedure that has been utilized in our clinic.

FOUR CASE REPORTS OF POST-TRAUMATIC CEREBRO-SPINAL FLUID RHINORRHOEA

The first two cases to be reported followed (*Case I*) a long series of frontal sinus operations in the course of which it is presumed an opening was made inadvertently in the posterior plate of the frontal sinus, the dura and the arachnoid, and the second case (*Case II*) followed a fracture of the anterior fossa of the skull 6 years before repair of the CSF leak was accomplished surgically; this was carried out several months after a severe attack of meningitis.

CASE I: F. M., white male, aged 38, was admitted to the Medical College of Virginia Hospital on the 14th February, 1948. His chief complaint on admission was increasingly severe convulsive seizures. He had had twenty-four separate operative procedures in his right frontal region during the previous nine years before admission to the neurosurgical service, involving, for the most part, the removal of sequestra and other sequelae of an initial right-sided purulent fronto-ethmoiditis (Fig. 1). Six years before admission, generalized convulsive seizures began, presumably due to scarring of the right frontal lobe and they were becoming more and more frequent. The bony decompression (incident to the numerous operations in the right frontal region) would protrude for a day or two after the seizure. For six years before admission he had had several episodes of rhinorrhoea from the right nostril followed by acute fulminating meningitis responding promptly on each occasion to sulfa drugs and penicillin. Undoubtedly before the introduction of these drugs the patient would have died in one of these very severe attacks of basilar meningitis. The last such infection had been two months before admission. The primary reason for admitting him to the hospital was for surgical repair and closure of the presumed cerebrospinal fluid fistula between the basi-frontal subarachnoid space in the right frontal region and the fronto-ethmoid sinuses. Sulfadiazine and penicillin therapy was begun forty-eight hours before operation. Except for poor vision in the right eye, he had no other complaints. Examination disclosed several scars in the right frontal and supraorbital regions. There was a bony defect about 7 x 3 cms. in the right frontal bone with deformity of the supraorbital ridge (Fig. 2). The skin was retracted and concave over this area except when the patient leaned forward at which time it protruded outward. There was no papilledema in either fundus and neurologically he was otherwise entirely normal.

Fig. 1. *Case I.* Appearance of patient before repair of cerebrospinal fluid leak from right nostril; 24 operative procedures in right frontal region for sinus infection before leak of fluid repaired.

Fig. 2. *Case I.* P-A skull and facial film to show defect in right frontal bone (arrows) incident to many previous operations for sinus infection. The communicating fistula in this case was in the region of the cribriform plate.

On the 18th of February, 1948, in collaboration with Dr. George Thrift, of the Otolaryngology Department, the cerebrospinal fluid leak was attacked through the old right frontal defect (JMM). Endotracheal ether anesthesia was utilized. The approach was through the scarred scalp area immediately above the remnants of the right frontal sinus. Normal brain tissue in the right frontal lobe was not seen at any time a ter the scalp was opened; the dura was non-existent under the skull defect. The posterior plate of the right frontal sinus was exposed after devitalized brain tissue from the right frontal lobe was aspirated with the suction apparatus. In the region of the cribriform plate to the right of the midline could be seen fluid leaking down into, and air from the ethmoids bubbling upward in a stoma several millimeters in diameter. No probe or curette was introduced for fear of making the opening larger and its occlusion more difficult surgically. The fistula intradurally was covered with several stamps of muscle which had previously been obtained from the left thigh. A strip of fascia lata was placed over the muscle, the entire area was covered temporarily with cotton and aspirated completely dry with the suction apparatus, after which no air or cerebrospinal fluid was seen in the opening; a water-tight closure of the fistula seemed to result from this procedure. Closure of the scalp was made with interrupted silk sutures without drainage. No remnant of dura could be found due to the many operations that had occurred previous to the intradural operative attack on the fistula itself.

Recovery was uneventful; the patient left the hospital on the ninth post-operative day. There had been no leak of CSF since the operation and his temperature had been normal for at least a week before discharge from the hospital. Phenobarbital was administered in moderate doses post-operatively. A recent letter from the patient in answer to our inquiry (March 12th, 1953), slightly more than five years after operation, stated that he had had an occasional convulsion and still requires penicillin at intervals presumably for residual sinus infection. He made no mention of any further attacks of meningitis. He has been working practically all the time since operation. He describes a "discharge" occasionally from his nose, which is probably mucus from the residual sinus infection. The patient has not been seen by us since operation, so it is not known for certain whether he is taking phenobarbital or other anti-convulsant drugs regularly at this time.

CASE II: W. B., colored male, aged 36, was first admitted to St. Philip's Hospital (M.C.V.) on the Medical Service on September 18th, 1951, with a diagnosis of pneumococcic meningitis. He had a history at that time of a CSF leak of fluid from the right nostril at intervals for the previous six years following a head injury, at which time he was struck across the forehead with a tree limb. He particularly noticed the fluid draining from his nose when he leaned forward; it would occasionally drain for 2 - 3 days and then stop spontaneously. A neurosurgical consultation on October 6th, 1951, after the pneumococcic meningitis was under control, recommended surgical repair of the fistula at a somewhat later date as soon as he had convalesced entirely from the meningitis. A spinal puncture at the time of admission (September 18th) yielded an initial pressure of 410 mm. of water, cloudy fluid, 4,060 WBC per cu. mm., 92% of which were polys. Two days later (September 20th) the fluid was again described as cloudy at the time of the second lumbar puncture (10,985 WBC's). Culture of the first spinal fluid obtained was reported as showing, twenty-four hours later, diplococcus pneumoniae. The patient responded rapidly to treatment with aqueous penicillin intramuscularly and terramycin intravenously and was discharged twenty days after admission (October 8th) with a normal temperature for the previous six days, all antibiotics having been discontinued for several days before discharge. Skull films, including special paranasal sinus films, made during this hospital period revealed old fracture lines in the basi-frontal region on the right side (Fig. 3).

Fig. 3. *Case II.* Re-touched P-A skull film showing (arrow) multiple fracture lines in the right inferior fronto-ethmoid region, the site of the fistula repaired at operation.

The patient was re-admitted on December 2nd, 1951. for operative attack on the presumed post-traumatic fistula connecting the basi-frontal-ethmoid sinus area on the right side with the supra-adjacent subarachnoid space. It was learned at that time that two and a half weeks after the head trauma had been sustained (six years previously), cerebrospinal fluid had begun to leak from the right nostril and had continued rather constantly ever since, there never having been a period of more than three consecutive days when there was freedom from CSF rhinorrhoea. His temperature, pulse and respiration at the time of the last admission were normal and his general

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condition was excellent, approximately two months after recovery from the pneumococcic meningitis with the aid of antibiotics. On 4th December, 1951, a right frontal osteoplastic flap was turned down (J.K.) and an intradural occlusion of the connecting fistula carried out with the aid of a muscle implant. The crista galli was indentified intradurally, and in the area of the olfactory bulb the first cranial nerve, which appeared to be composed largely of degenerated nerve fibers, was gently avulsed and a large muscle implant inserted firmly in the olfactory groove and cribriform plate area and firmly stamped down by strong suction over cotton and downward pressure by the operator's finger for a minute or two. It is of interest that in spite of a degenerated-appearing first cranial nerve on the right side, the sense of smell (to peppermint) was present in the right nostril before operation. The dura in the frontal region behind the frontal sinus showed an inflammatory reaction, probably attributable to the recent meningitis. The dura was tightly closed without drainage and the bone flap replaced. The patient's temperature was elevated to 102°-103° on several occasions in the first six post-operative days but fell thereafter abruptly and remained normal until discharge on the twelfth post-operative day. At that time, the patient was in excellent condition and the area of operation in the right frontal region was completely healed. In the succeding fifteen months that have elapsed since operation to the date of this report he has remained well with little or no headache and there has been no leak of fluid from the nose. In two post-operative visits to the out-patient clinic, he has been asymptomatic and has remained so. On March 11th, 1953, the patient wrote in answer to our inquiry that there was no leak of fluid from his nose since the operation: "I am working and getting along fine."

Cases III and IV are, on the other hand, both due to *gunshot wounds, Case IV* being further complicated by pneumocephalus involving the ventricles, meningitis, bilateral choked disks and subarachnoid hemorrhage, received in the hospital as a *delayed* case.

CASE III: A. H., white male, aged 41, was admitted to the Medical College of Virginia Hospital on the 8th of January, 1952, in a critical condition immediately after a gunshot wound of the frontal portion of the head involving both cerebral hemispheres by a .32 caliber pistol bullet. It has never been definitely determined whether the bullet wound was self-inflicted of homicidal. The wound of entrance was in the right inferolateral frontal area of the head. From this point, the bullet (as shown by X-ray films made at the time of admission) travelled, in a transverse trajectory across the right frontal lobe, crossed the midline either through or below the falx cerebri to lodge in the left frontal lobe just beneath the dura, above and to the left of the orbital rim of the left orbit (Fig. 4). He had not been definitely rendered unconscious by the injury, had walked away from the scene of its infliction and was talking and cooperative at

Fig. 4. *Case III.* P-A skull film at time of first admission. The wound of entrance is on the right; the direction of the bullet was from right to left; the bullet (.32 caliber) is seen lying superficially in left frontal region. Note multiple fracture lines involving sinuses.

the time of admission, several hours afterward, although his responses to questions were obviously not reliable. The X-ray films of the skull, in addition to showing the course of the bullet already referred to, also showed multiple fracture lines in the frontal sinus-ethmoid area, bilaterally. Although he could see with each eye when admitted several hours after the injury, within one and a half hours after admission to the hospital, marked amblyopia had developed in the right eye. It was thought that the course of the bullet had been very close, probably just superior, to both optic nerves.

The patient was operated upon (JMM) on the day of admission, primarily (1) to debride the wound of entrance in the right frontal area and thus to prevent a fatal cerebritis, and (2) to remove the bullet if it could be quickly found beneath the intact skull in the inferior left frontal area. These procedures were carried out without great difficulty or loss of blood, the patient being under intravenous pentothal and endotracheal gas-oxygen-ether anesthesia. Pulpified brain was oozing from beneath the torn dura when the wound of entrance in the right frontal area was debrided. The bony injury had penetrated the right frontal sinus (Fig. 5) and gelfoam was placed in this structure. Bleeding from the cerebral cortex and

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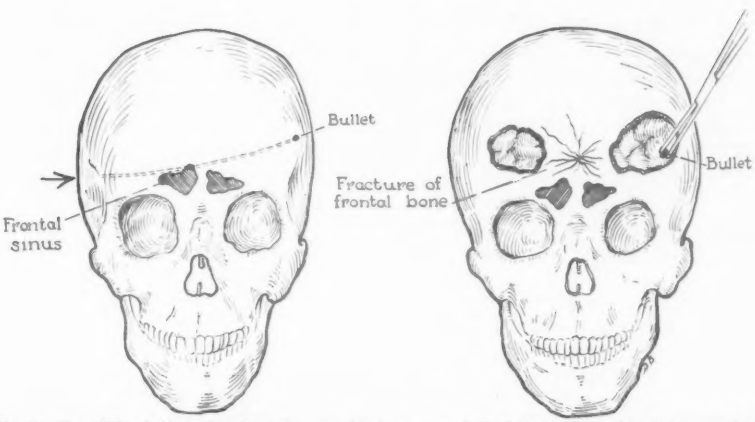

Fig. 5. *Case III.* **Artist's drawing of approximate course of the bullet. Note that it traversed the frontal sinus on right side, thus accounting for the cerebrospinal fluid rhinorrhoea repaired at later date by intradural muscle implant. View on right shows extraction of the bullet in left frontal region through a separate craniectomy.**

subjacent white matter of the right frontal lobe was controlled with the electro-surgical cautery and warm saline irrigations. No drainage was employed in either wound. The bullet was located in the left lateral-inferior frontal area by measuring its location on the skull films and then on the patient's shaved scalp. By means of a small craniectomy in that area the bullet was readily found subdurally and removed by forceps. The dura was widely opened because of cerebral edema and gelfoam placed over the brain. The scalp was tightly closed in layers without drainage. The next day the patient could count fingers with each eye and intraorbital pressure was not excessive. Nine days later (January 17th) a secondary closure of the left frontal wound was carried out as the marked edema of the left frontal lobe had caused a separation of the scalp wound. Under endotracheal ether anesthesia the old incision was re-opened and additional brain tissue aspirated away and several cc. of a weak penicillin solution (5000 u/cc.) were instilled in the cerebral wound. The galea was approximated with interrupted sutures of catgut and the skin with silk. No drainage was employed. The wound healed satisfactorily and caused no further difficulty. After a rather stormy convalescence, the patient was discharged approximately six weeks after the initial injury, his discharge from the hospital being delayed by a not unexpected "frontal-lobe syndrome" characterized for several weeks by marked restlessness, fabrication, euphoria, urinary incontinence and poor cooperation.

He continued to have a "moist" right nostril from the date of discharge (February 23rd) after the two original operations until re-admission (May 18th) for the insertion of two tantalum plates in the two frontal bony defects (Fig. 6) and a muscle implant in the opening in the posterior plate of the right frontal sinus which had resulted from the original bullet wound, which in turn had been followed by a rhinorrhoea on the right side. On the right side, the frontal lobe remnant was elevated (May 20th) and a 2 mm. fistula found extending through the posterior wall of the right frontal sinus. Muscle was inserted tightly intradurally in this opening, the scalp

Fig. 6. *Case III.* **Post-operative P-A view of skull showing two small separate craniectomies, the one on the right the debrided wound of entrance, that on the left, the site of the bullet removal. These bony defects were repaired later by small tantalum plates (see text).**

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closed without drainage and he has not leaked fluid from the nose since then, ten months after the repair of the CSF fistula. He remains in excellent condition at this time, as reported to us by letter (March 14th, 1953) and also has been free of headaches and other ill effects since his last operation: "My eyesight has fully recovered. I am starting a new business next month."

CASE IV: Dr. L. R., aged 48, a white male physician was admitted to the Medical College of Virginia Hospital on April 19th, 1952, in a critical condition following a self-inflicted gunshot wound of the head involving both cerebral hemispheres ten days previously at his home in another state (.45 caliber pistol bullet fired through a rifle into the roof of his mouth). He was not rendered unconscious by the bullet and was found standing after the shot in his examining room by an entering patient. The bullet was lodged in the left parietal area as seen readily on the X-ray films and the wound of entrance was in the hard palate just to the right of the midline. Skull films sent with the patient to the hospital made on the day of injury showed only these findings (Fig. 7). At the time of

Fig. 7. *Case IV.* Forty-five caliber pistol bullet lodged in parietal region just to left of midline, as seen in lateral skull film made immediately after injury. The bullet entered the intracranial cavity through the mouth and the right basi-frontal region, ricochetting eventually into the left parietal area.

admission, he was conscious and fairly rational although childish and very poorly oriented as to time and place. There was a leak of spinal fluid from the right nostril. He had been placed on antibiotics at home immediately after the injury but had complained of increasingly severe headache in the right frontotemporal region. Four days after the injury a spinal puncture—done elsewhere—showed an initial pressure of 540 m. water and 1,000,000 RBC per cu. mm. indicative of a high degree of increased intracranial pressure and rather marked subarachnoid hemorrhage. Papilledema developed not unexpectedly with these complications and when seen by us, ten days after the bullet wound was inflicted, he had 2-3 D. elevation of each optic disk. He was ambulatory but not very cooperative. Temperature was 101.6 rectally. Vision seemed good in each eye. The visual fields were grossly normal by confrontation test. There was mild hypertonicity in the right leg, the bullet being in the left parietal lobe near the vertex of the head. It was assumed that the bullet had entered the right basifrontal area of the intracranial cavity through the roof of the mouth, traversed the right frontal lobe obliquely from before backward, penetrated the falx and had come to lodge just beneath the dura, probably, slightly to the left of the midline in the parietal area. Because of the persistence of the headache, skull films were repeated (April 21st, twelve days after injury) and a perfect image of the ventricular system was seen, i.e., air had undoubtedly entered from the frontal sinus-ethmoid area on the right side, traversed the severely traumatized right frontal lobe and entered the anterior horn of the right lateral ventricle, during coughing or straining, to produce a typical ventriculogram. (Fig. 8).

Fig. 8. *Case IV.* Right lateral skull film made twelve days after injury showing perfect image of lateral ventricles (spontaneous ventriculogram) and separate rather large extra-ventricular air pockets in the right frontal lobe.

In summary, then, here was a severely lacerated right frontal lobe, a .45 pistol bullet in the left parietal lobe, a CSF fistula with rhinorrhoea in the right basi-frontal area, which included a direct connection with the ventricular system (aerocele), severe subarachnoid hemorrhage, a high degree of increased intracranial pressure with choked disks bilaterally, and undoubtedly a considerable degree of basilar meningitis and right cerebral cerebritis, first seen by us *ten days* after trauma. A considerable problem in surgical judgment presented itself as to the best procedure to undertake in surgically attacking these lesions and *when* to do so. As the patient had considerable fever on admission, and was not drowsy or stuporous, it was decided to ignore the bullet (rarely the major acute problem in bullet wounds of the head compared to retained bone fragments in the brain when the bullet is lodged at the end of a long trajectory in the brain), and to treat the patient vigorously with antibiotics for a few days, at least, in an effort to eliminate the meningitis but to operate as soon as possible due to the ventricular-subarachnoid-fronto-ethmoid fistula and CSF rhinorrhoea associated with bilateral choked disks. This plan was followed and nine days later (19 days after injury), the TPR then being entirely normal, a large right frontal bone flap was outlined and turned down (April 28th). A left frontal burr opening had been

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made just previously and the anterior horn of the left frontal lobe tapped to be certain that there was no left-sided subdural hematoma or intracerebral abscess. The left anterior horn was tapped and 60 cc. of clear ventricular fluid obtained thus eliminating any mass lesion in time of this report, approximately eleven months after the wound was sustained.

SUMMARY

(1) Post-traumatic CSF rhinorrhoea with or with-

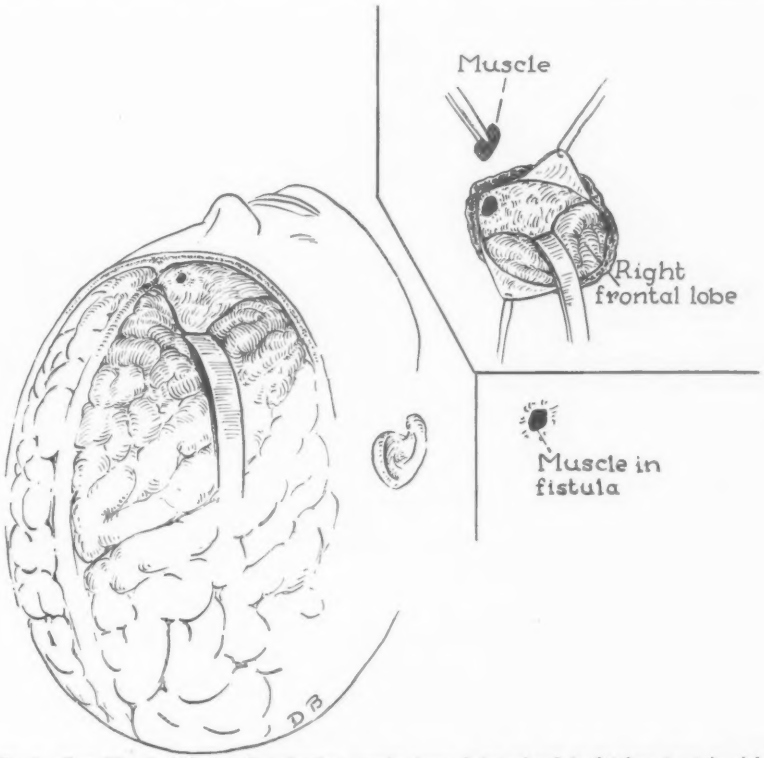

Fig. 9. *Case IV.* Artist's drawing showing muscle stamp being placed in fistulous tract in right cribriform plate, after its exposure under retracted and elevated right frontal lobe. This is essentially the method employed in all four cases herein reported.

this area. The right-sided frontal bone flap was then turned down (JMM) and discarded as the brain was quite full and very over-vascularized (? cerebritis) in spite of the left anterior horn having been tapped twice immediately before craniotomy. A ventricular needle in the right frontal lobe was followed by a "puff" of air several cm. from the surface; the frontal pole of that hemisphere was then elevated and muscle stamps were placed over the fractured site in the right cribriform plate (Fig. 9). The scalp was tightly closed without drainage and the patient has remained well since, except for his mental status. On May 10th, 1952, (12 days after operation) skull films showed no air in the ventricles (Fig. 10). When seen in the office slightly more than 6 months after operation (November 4th, 1952), his general condition was excellent, the optic disks were normal, and no CSF leak had occurred from the nose since operation. He was ambulatory and had gained at least fifteen pounds in weight. He is still confined to a mental institution at the

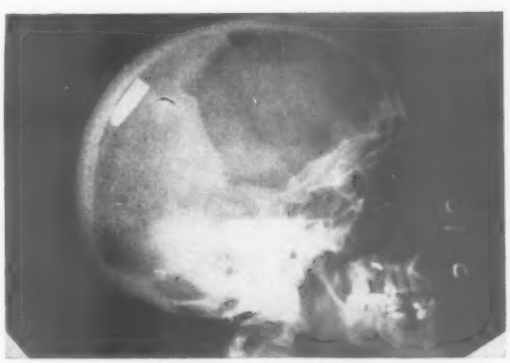

Fig. 10. *Case IV.* Lateral skull film made twelve days after repair of CSF leak through right nostril and tapping of lateral ventricle and both frontal lobes with release of entrapped air. Note (1) absence of bone flap due to cerebritis and edema, and particularly (2) absence of ventricular or frontal lobe air (compare with Fig. 8.) The bullet is still *in situ*, adjoining the longitudinal sinus. The patient remains in excellent condition thirteen months after injury and operation.

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out complicating pneumocephalus is discussed with particular reference to the recognition of the condition and the satisfactory non-operative and operative management of this serious lesion.

(2) Four illustrative cases (two of them due to gunshot wounds) from our clinic are presented—all operated upon by a uniform intradural method and occlusion of the fistula by muscle with successful outcome in each instance, to date.

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PR TIP OF THE MONTH—The patient who drives 'round the block for twenty minutes before she can find a parking place near the doctor's office—or who receives a parking ticket because the doctor was late in seeing her often complains loudly about doctors. Consequently, many physicians and medical societies are making *special parking arrangements* for the convenience of such persons. It makes good public relations sense.

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UNUSUAL COMPLICATIONS IN DIABETES MELLITUS—CASE REPORT

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There have been several reports in the literature concerning cystitis emphysematosa and necrotizing renal papillitis occurring in diabetics; and many articles on cortical necrosis due to a variety of causes, but to our knowledge this is the first report of all three conditions occurring in the same patient.

Case Report

Mrs. B. D. W. (123059), a 57 year old white housewife and a known diabetic of 6 years, was admitted on November 3, 1951, with a chief complaint of pain in the left flank, side and lower abdomen of 24 hours duration. The pain had come on suddenly, was persistent, sharp, and localized. There was associated nausea, but no vomiting, diarrhea, nor melena. She stated that she had been passing gross blood in her urine for twenty-four hours and had more burning, and frequency, up to ten times at night, than usual, having had these latter symptoms since 1946.

Her past medical history was significant in that she was known to have had diabetes mellitus for 6 years, which had proved difficult to control even with insulin and a rigid diet. She had lobular pneumonia in 1942 without sequelae, and malaria as a child. In February, 1946, she had a right nephrectomy for a staghorn calculus. In September, 1946, she had a vaginal hysterectomy and perineal repair because of prolapse of the uterus.

Laboratory studies in September, 1946, showed: Cath. urine: sp.gr. 1.014, reaction acid, albumin trace, sugar 4-plus; microscopic: numerous wbc, and numerous rbc per h.p.f. A urine culture at this time grew out E. coli. Blood sugar ranged from 305-130 mgm. %. Blood urea was 52 mgm. %. A KUB did not reveal any suspicious shadows.

The review of systems and family history were non-contributory.

Physical examination on the present admission revealed a pale, stout, elderly, white female with drawn facies, complaining of severe pain. Her temperature, pulse, and respirations were 102 F., 120, 22, respectively. Her blood pressure was 118/78. The tongue was dry, and there was an acetone odor to her breath. Fine moist rales were heard over both lung fields. The cardiac rate was increased and a systolic murmur was present. Moderate tenderness with spasm was present over the left costovertebral angle and left lower abdominal quadrant.

Laboratory data was as follows: Urine: sp. gr. 1.014, acid reaction, 4 plus albumin, 3 plus sugar, and numerous rbc/hpf. No acetone nor diacetic acid was present. Urine culture grew out E. coli. The blood count was normal. Her blood sugar was 302 mgm. %, and blood urea 68 mgm. % on admission.

The patient's diabetes was treated by a strict diabetic diet, parenteral fluids and 60 U NPH insulin supplemented with regular insulin. She was also given antibiotics, analgesics, digitoxin, and oxygen. Her prognosis was poor. Urinary output was diminished. On November 5, 1951, cystoscopy was performed to find if the solitary left kidney was blocked. A small amount of urine was obtained. The bladder mucosa was intensely red throughout. The lateral walls and trigone were profusely studded with crepitant air blebs beneath the mucosa. The ureteral orifices could not be located. The patient tolerated the procedure well. She was given vigorous supportive treatment the next four days, but she continued to go downhill, became comatose, and expired on November 9, 1951.

At autopsy (Dr. M. B. Beecroft, Pathologist) massive cortical necrosis and necrotizing renal papillitis of the left kidney were found. The microscopic picture of these lesions has been well described by Edmondson, Martin, Evans and Robbins, Mallory and Kinney, and others, and will not be repeated here. Glomerulosclerosis was not observed. There were air blebs in the submucosa of the bladder. A totally unexpected finding was a ruptured dissecting aneurysm of the left common iliac artery with massive retroperitoneal hemorrhage, the immediate cause of death.

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DISCUSSION

There has been much speculation as to the etiology of cystitis emphysematosa. The possible causative factors have been divided into four classes: (1) Anærobic gas-forming bacteria, which produce a generalized virulent infection, may cause an associated cystitis emphysematosa. (2) Cystitis emphysematosa may develop as a result of invasion of the mucosa and submucosa of the bladder by a gas-forming bacillus which also produces a hemorrhagic cystitis. (3) An abacterial cystitis emphysematosa is found in diabetics with a hyperglycemia of 200 mgm. % or more; the gas is supposedly formed by enzymic action in the mucosa and submucosa of the bladder. (4) Those cases of cystitis emphysematosa that result from abrasions (catheters, cystoscopes, or other instrumentations), in which the air or gas-producing bacteria are introduced under the epithelium. It is interesting to note that in our case there is a possibility that three of the above etiological factors may have played a role: Our patient's blood sugar level was over 300 mgm. %; a colon bacillus was cultured from the urine; and there was a chance of the introduction of air or gas-producing bacteria during catheterizations or cystoscopy.

The finding of necrotizing renal papillitis at autopsy was a not unexpected observation. This is a severe, usually fatal, type of acute pyelonephritis, distinguished by necrosis of the renal papillae, which occurs in middle-aged diabetic patients, and also in cases of severe urinary tract infection with obstruction but without diabetes. The criteria for the clinical diagnosis of N.R.P. in diabetic patients has been given as fever, hematuria, renal colic, unexplained coma, and sudden increase in severity of symptoms of known pyelonephritis—all of which were found in this case. Slipyan and Barland present a stimulating discussion of possible etiological and anatomical basis for this lesion. Pyelographic studies were not done but might have demonstrated the irregular filling defects of the renal calices due to necrosis, and sloughing of the tips of the papillae. Early diagnosis is essential in order to interrupt the process of infection before it progresses to the stage of necrosis.

Cortical necrosis has been associated with a wide variety of conditions. Most authors agree that necrosis of the renal cortices is ischemic in origin whether it be due to intense vaso-constriction, vaso-paralysis, thrombosis or necrosis of the arteries by-passed.

The work of Homer Smith, Trueta, Goodwin, Sloan, and Scott on this problem has been basic. The latter investigators confirm the work of Trueta, and conclude that there is a neurovascular control of the renal blood flow and that renal and/or splanchnic nerve stimulation may permit the juxta-medullary glomeruli to carry the blood through the kidneys, by-passing the cortical glomeruli which become ischemic. Goodwin, Cason, and Scott state that their experiments would seem to show that "the triad of shock, splanchnic nerve stimulation, and hemoglobinemia will produce the lesion clinically and pathologically, whereas anyone of the three taken separately or in pairs will not". Trueta also found that any intense injury or insult to the iliac vessels can set up a neurovascular reflex that may result in cortical necrosis of the kidneys.

The clinical application of this neurovascular reflex has been well demonstrated in cases of transurethral resections of the prostate, rough handling of the ureters, following cystoscopy, over-whelming abdominal surgery, shock, crush injuries, vascular injuries, etc. In our case, in addition to the cystoscopic examination as a possible contributory factor, the autopsy revealed the presence of dissecting aneurysm of the left common iliac artery.

SUMMARY

A case of diabetes mellitus with cystitis emphysematosa diagnosed ante-mortem is presented, which, in spite of intensive therapy, succumbed rapidly. At necropsy a necrotizing renal papillitis, cortical necrosis of the kidney, and a dissecting aneurysm of the left common iliac artery with massive retroperitoneal hemorrhage were found. These findings again demonstrate the need to be alert for unusual complications in individuals with diabetes mellitus and urinary tract infection, and the value of postmortem examinations in diabetics even though the cause of death may appear to be obvious.

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Home Care Advantageous Over Hospitalization in Some Cases.

Home care for the chronically ill and for children with chronic active rheumatic fever has many advantages over hospitalization, two articles in a recent J.A.M.A. disclosed.

A 30-month study of 45 children with rheumatic fever showed that "when certain initial criteria for home suitability are met, most children with active rheumatic fever and free of congestive heart failure can be cared for as well at home as in the hospital, with an equally good end-result and with greater over-all advantage to the patient and his family." The study was made by Drs. Dennison Young, White Plains, N.Y., and Manuel Rodstein, New York City, who are associated with the division of social medicine and the medical division of Montefiore Hospital.

Dr. Louis Udell, Philadelphia, described a 39-month study of 160 chronically ill persons who were treated at home. Results indicated that "those who display readiness to cooperate in intensive care show improvement physically, emotionally or socially in situations formerly believed static or hopeless." . . . "Many patients who do poorly in the hospital do much better in their home surroundings, eating the food that suits their palate, seeing their friends at anytime, and being with their loved ones."

The cost of the care of the patient at home is much less than the cost of caring for the patient in the hospital, Dr. Udell stated. In addition, home care will help to alleviate the shortage of hospital beds.

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CLINICAL ASPECTS OF BARBITURATE INTOXICATION

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The problems of barbiturate addiction and suicide attempts with barbiturates are encountered with increasing frequency. The extent of the addiction problem was forcefully brought to the attention of one of the authors during a year that he was attached to the staff of the U. S. Public Health Service Hospital at Lexington, Kentucky. Though a hospital for narcotic addicts, many of the patients admitted there also had either active barbiturate habits or had used barbiturate drugs extensively in the past. Approximately one out of twenty addicts to narcotics are started on the road to addiction by medication prescribed by a physician. However, a much larger percentage of barbiturate addicts are given their start by injudicious prescription of these drugs. As little as a half gram of a barbiturate daily may be an addicting dose, though of course this varies depending on individual tolerance. Practically every new patient we see is taking barbiturates to some extent.

The frequency of suicide attempt by barbiturates is increasing by leaps and bounds each year, although the over-all suicide rate has declined. Barbiturates are used more often than any other poison in attempting suicide, as any physician with experience in the emergency room of a general hospital will recognize.

We feel that it is the responsibility of all physicians and those working in allied professional fields to recognize the dangers of barbiturates and the results of making these drugs available to patients without adequate restriction. We see two definite types of risks—suicide and addiction.

In chronic barbiturate intoxication, as seen in addiction, there are symptoms of depression of the cortex. These include drowsiness, confusion, inability to concentrate, and impairment of judgment. In addition the barbiturate addict presents signs of dysfunction of the cerebellum with ataxia, nystagmus, slurred speech, tremors, adiadokokinesis, and choreiform movements. It is obvious that many of these signs and symptoms are similar to those seen in chronic alcoholics.

Barbiturate addicts are subject to injuries due to falling, they may be involved in automobile accidents while under the influence of these drugs, often smoke in bed and cause fires, and may even commit criminal acts. These addicts tend to become disturbed in their inter-personal relations and even to become social outcasts as far as their families, old friends, and employers or fellow employees are concerned. Barbiturates are often prescribed to help emotional disturbances and yet when used excessively and unwisely by the patient, they may provoke severe depressions, emotional lability, infantile behavior, aggressive acts and paranoid ideation.

Upon withdrawal from any active barbiturate habit, a patient rapidly develops fatigue, anxiety or apprehension, fasciculation and coarse tremors of the hands and face, especially peri-oral. Later the patient will suffer abdominal cramps, nausea and vomiting, weight loss, and rather severe insomnia. The pulse, temperature, and blood pressure are all elevated. Some 2½ days after withdrawal the patient is apt to have one or more grand mal convulsive seizures and some 3 to 7 days after withdrawal a psychosis may develop. Characteristically this barbiturate withdrawal psychosis resembles the delirium seen in alcoholic psychosis; however, it is related to the withdrawal of barbiturates and is relieved by the administration of barbiturates, whereas this is not true in the case of alcoholic psychosis. Most psychoses following the withdrawal of barbiturates will clear in less than two weeks but some may go on for a longer time, even for two or three months, and in that event they often resemble more nearly a schizophrenic reaction.

Acute barbiturate intoxication as seen in accidental over-dosage or suicide attempts is characterized by unconsciousness with slow, shallow respirations and cyanosis. The history obtained from reliable informants aids in the diagnosis. The laboratory can help us also in diagnosing cases of barbiturate coma.

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The State Toxicology Laboratory can analyze gastric contents or specimens of blood or urine and give us a report within 30 to 45 minutes during the day and 1 to 1½ hours during the night.

In this paper we will review our experiences with acute and chronic barbiturate intoxication at the Medical College of Virginia Hospital Psychiatric Service during the year 1952. This includes (with exceptions noted later) only patients admitted to the Psychiatric Service, though many more were seen in the emergency rooms and handled through admission to another hospital or referral to the out-patient clinic. Some who were seen in the emergency rooms were taken home against medical advice by relatives who did not appreciate the severity of the problem.

Of a total of 747 in-patients treated by the Psychiatric Service during 1952, 32 patients had barbiturate drugs as a distinct part of their problem. This represents a rate of 4.3%.

Of these 32 patients, 19 were admitted to Psychiatry actively addicted to barbiturates or had used them in addicting quantities previously; 13 patients were admitted for barbiturate over-dosage in attempted suicide. It is of note that 4 doctors and 1 nurse were among the 32 patients.

Of the 19 patients admitted for addiction, 5 either had on admision or subsequently developed, during their hospital stay, withdrawal psychoses. One of these patients had a history of withdrawal psychosis which had cleared prior to admission. Ten patients had barbiturate withdrawal convulsive seizures after admission or prior to admission. Four patients of this group had both barbiturate withdrawal convulsive seizures and psychotic episodes. There were no deaths among this group. Our treatment for these addicts included stabilization on an adequate dosage sufficient to produce mild intoxication and then gradual withdrawal from barbiturates usually proceeding at the rate of reduction of 1½ grains every one to two days. These patients must be watched closely from the standpoint of impending convulsive seizures and this often means side rails on the bed, proper escort to the bathroom, etc. When indicated, psychotherapy was a part of our treatment, though unfortunately many addicts are fixed at such an infantile level that this type of therapy does not have much to offer. In addition, many of these patients stayed with us during the period of reduction but not long enough for any intensive psychotherapy in a hospital setting.

The problems encountered in withdrawing a patient from barbiturates may readily by illustrated by the following case report:

One of the patients in our addict group was a 45-year old white female using barbiturates for several years following her husband's death. The patient had previously been treated in other hospitals for addiction but had never managed to stay away from drugs for any length of time. She was admitted to our Psychiatric Service actively addicted to barbiturates, the dosage being estimated by the patient and relatives as 30-33 grains of Nembutal per day. In spite of stabilization on an adequate dosage, and careful and gradual withdrawal, she subsequently developed a barbiturate withdrawal psychosis four days after admission—with confusion, disorientation, delusions, and auditory and visual hallucinations. She was maintained on optimum barbiturate dosage during her psychotic period and in addition received ACTH. Her psychotic episode cleared after three days, or about the seventh hospital day, and her course thereafter was uneventful. Completion of her withdrawal was accomplished at our hospital and a convalescent nursing home to which she was transferred for financial reasons. Unfortunately, as is the case with so many addicts, she returned to our hospital a few months later with another roaring habit and was then committed to the state hospital for treatment.

As will be recalled, of our 32 patients admitted with a definite problem in regard to barbiturates, 13 of these patients were admitted for barbiturate intoxication after a suicide attempt. Of these 13 patients, ten were diagnosed re-active depressions, two involutional depressions, and one undiagnosed. The majority of these patients received the usual treatment with picrotoxin or other drugs in the emergency room. One patient of this group was revived with the aid of the Reiter Electro Stimulator, and we will speak more of this machine later in this paper.

Some patients, of course, take so few capsules that heroic measures in the emergency room, etc., are not necessary. However, our experiences indicate that even patients who take a few capsules may have made a full-fledged suicide attempt, and that their use of an obviously innocuous dose cannot be dismissed as a suicidal gesture. It is a policy at our hospital that Psychiatry sees every case of barbiturate intoxication. One patient who took only a

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few capsules of one of the barbiturates was signed out of the emergency room by a responsible party only to jump off a bridge the same day. One other patient took a few barbiturate capsules and also turned on the gas, indicating her definite intention to do away with herself. A third patient, in addition to taking barbiturates, had inflicted severe lacerations, emphasizing her real intent. Of course, lay people are not schooled in the lethal dose of barbiturates and may take a few capsules, thinking that this will be sufficient. It certainly behooves us to do our utmost not to provide great amounts of barbiturates for patients with self-destructive tendencies.

At the Medical College of Virginia Hospital we have a Reiter Electro Stimulator available for the treatment of patients with acute barbiturate intoxication. Of our group of 13 patients admitted to Psychiatry during 1952 for a suicide attempt with barbiturates, only one received Reiter treatment. We also gave this type of treatment to one of the patients in the addict group admitted in an unconscious condition, though it could never be determined definitely whether this represented actual over-dosage in a suicidal attempt. Two other patients not admitted to the Psychiatric Service received Reiter Electro Stimulation during 1952 at our hospital for barbiturate over-dosage, in the emergency room.

One of these was fully aroused from her barbiturate coma with Reiter treatment but died three days later of the toxic effects of another poison she had taken in conjunction with the barbiturates. The second patient not admitted to our service but receiving Reiter treatment for barbiturate over-dosage was a Negro nurse who had taken an allegedly massive over-dosage of barbiturates and could not be roused adequately with other chemical means. When Reiter Stimulation was begun patient was areflexic and in shock, but after 93 minutes of continuous Reiter treatment she was roused and was talking. Two days later, while the patient was awaiting transfer to a state hospital following commitment procedures, a bottle of 100 phenobarbital tablets was found in her bed. She had appropriated these during her brief hospital stay from a locked cabinet somehow. We should also like to call attention to one patient treated during 1953 with the Reiter machine. This patient, a nurse, ingested 31½ grains of Seconal in a suicide attempt and after admission to the Psychiatric Service was areflexic and in shock. She was revived with the help of Reiter Electro Stimulation maintained almost continually for a period of twelve hours.

It seems in order at this point to mention in more detail the Reiter apparatus which we feel to be a valuable aid in the treatment of acute barbiturate intoxication, whether this is the result of suicidal attempt, accident, or over-dosage in the course of sedation of a patient. This machine provides continuous electrical stimulation over as many hours as are required to rouse the patient. By increasing the respiratory rate, it brings about a more rapid elimination of barbiturates and by an earlier awakening tends to reduce such complications as pneumonia, etc. We advocate its immediate use in all cases of profound barbiturate coma, though other measures such as gastric lavage, chemicals, and frequent shifting of the patient, should be used in addition to the Reiter treatment. We certainly think that the emergency room in any city hospital ought to be aware of what the Reiter machine can do for patients brought in unconscious from barbiturate overdosage.

In summary, we have reviewed some of the aspects of acute and chronic barbiturate intoxication in general, and have discussed patients on our Psychiatric Service in 1952 who had a definite problem with regard to barbiturates, either in the realm of addiction or over-dosage in suicide attempt. We would like to urge again that all members of the medical profession and allied fields consider the dangers involved in making barbiturate drugs easily available to patients.

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501 East Franklin Street

A CONCEPT OF MUCOVISCIDOSIS (CYSTIC FIBROSIS OF THE PANCREAS)*

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The clinical syndrome of mucoviscidosis, as well as various laboratory aids in diagnosis, has been well established, but the pathogenesis of this disease is still in a state of flux. Until this is established the disease cannot be well understood, and therefore, at present, remains inadequately treated. Basically there are three theories of pathogenesis which to date have not been refuted. These are, first, that it may be an infection in utero[2] or the result of a postnatal infection.[20] But, if so, the selective ability of the damaging organism is most remarkable. The second theory is that it is a genetic problem and it has rather conclusively been shown that it occurs in families in a manner consistent with a mendelian recessive trait. The frequent presence of other congenital defects in these children helps to lend support to the genetic origin.[2,3,4,20] The third theory was first expressed by Farber who believes that the disease is the result of the functional imbalance of the autonomic nervous system governing the secretion of mucus throughout the entire body with the result that the mucoprotein fraction of this mucus is abnormal. This in turn causes the characteristic disease picture seen in the several organ systems.[5,7,8]

From our study of mucoviscidosis, based on our own autopsy findings and of that recorded in the literature, we feel that the most plausible theory is that of widespread abnormal mucus production on a congenital basis, and that the disease process begins in the embryologic entodermal tube before it divides to form the respiratory and gastro-intestinal tracts. To our knowledge this concept has not heretofore been expressed. It seems reasonable that an alteration on a congenital basis of a glandular mucus enzyme system could also explain the production of abnormal mucus with its established disease pattern. The pathogenesis of the disease would then be as in Fig. 1.

This concept refutes the idea that the disease originates in the pancreas and somehow, in some mysterious manner, spreads to the other organs involved[19] and amplifies Farber's[5] theory of a systemic disorder. If then, one accepts the concept that the abnormality exists in the original entodermal tube, it is not difficult to explain how or why the disease also coexists in the several widely separated organs and with varying clinical pictures. Meconium ileus with resulting intestinal obstruction is characteristic of the disease in the early newborn period.[1] During infancy, cough, emphysema and recurrent pneumonias are statistically more prominent, but vomiting, and bulky, foul stools are also fairly frequent. The full blown picture of both lung and intestinal involvement is most typically seen in the early childhood period.[1,6,7] From autopsy reports we find that the extent of pancreatic involvement can also vary with reports of as little as 20% of the organ showing characteristic pathology.[13,20,15] The liver seems least often to be implicated.[13,14] It is fairly easy to conceive that when the two tracts form from the original entodermal tube, that any proportion of diseased glands may split off to follow either or both paths, giving us then these widely varying clinical findings and symptomatology.

At the Medical College of Virginia Hospitals five cases of mucoviscidosis have come to our attention, four of them autopsied and one diagnosed at surgery. The five case histories follow:

Case 1. Baby girl B. was a seven pound, fifteen ounce colored female infant delivered by low forceps, who vomited meconium stained material frequently from birth until operation after forty-eight hours of life. X-rays showed small bowel obstruction, and subsequent surgery revealed thick inspissated meconium in the terminal ileum, which was manually removed. No other pathology was found to account for the obstruction. The postoperative course was stormy and the child expired two days after surgery. Of interest is the fact that this child exhibited numerous other congenital anomalies. The hair on her head was kinky and snow white except for two circular patches approximately two inches in diameter in the occipital region, which were coal black in color. There was complete agenesis of the

*Read at the annual meeting of the Medical Society of Virginia in Richmond, September 28-October 1, 1952.

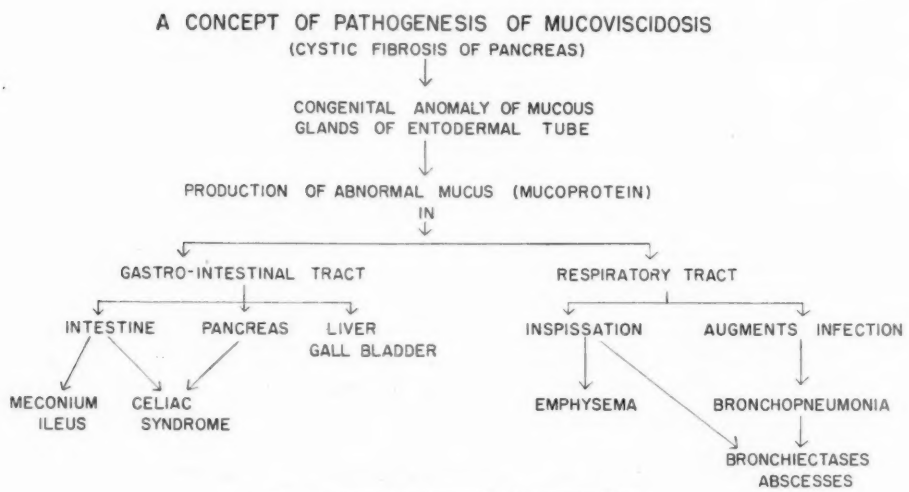

Fig. 1. Proposed pathogenesis of mucoviscidosis.

right pinna and external ear canal. Absence of pigmentation of the skin existed in a dermatome pattern over the arms, upper thorax, and lower extremities.

Case 2. M.G. was a four month old colored male, who had never done well, with frequent vomiting since birth. The last two months of his life he had many "head and chest colds" with a deep hacking cough which had become progressively worse. He was small and poorly nourished with an emphysematous chest and inspiratory and expiratory rales to auscultation. There was no response in his chest condition to all the usual forms of therapy. Toward the end of his life he developed huge, bulky stools accompanied by severe dehydration with electrolyte imbalance, and ceased, in spite of heroic treatment. At autopsy a widely patent foramen ovale was also found.

Case 3. V.M.W. who was a 2 month old white female, breast fed for the first month of life and who nursed eagerly, but was never satisfied. At one month of age her stools became frequent with a foul odor and she had been vomiting 5 days before hospital admission. She was somewhat edematous and the liver edge was palpable 3 finger breadths below the right costal margin. Autopsy revealed typical cystic and fibrotic changes in the pancreas with marked fatty changes in an enlarged liver. The lungs showed no pathology.

Case 4. F.D. was a 5 month old, white male, first admitted in a dehydrated, shock-like state at 4 months of age, because of vomiting and abnormal stools. He improved on a Mullsoy regime and was sent home only to return in two weeks with vomiting, abnormal stools, edema and with a liver edge felt at the iliac crest, expiring suddenly after 3 days. Post-mortem examination showed little, if any, involvement of the lungs. Typical cystic fibrosis of the pancreas existed. The huge liver was almost entirely replaced by fat and the bile ducts were also involved.

Case 5. M.A.R. was a seven year old white female with seven hospital admisisons during her brief life, the first because of constant hacking cough and marked weight loss at three years of age and a chest X-ray compatible with miliary tuberculosis and emphysema. When approximately four years old, large foul stools appeared with continuance of cough and rales in the chest. The chest was emphysematous and to X-ray showed extensive bilateral linear mottlings, suggesting bronchiectasis and interstitial pneumonitis. Decreased trypsin in the duodenal drainage was found at that time and the diagnosis of cystic fibrosis of the pancreas with lung involvement was established. She made a marked improvement on pancreatin granules and was supported with various antibiotics, but still returned with serious lung infections. On her last admisison she was cyanotic, dyspneic, with substernal and subcostal retraction, showed clubbing of her digits and evidence of heart failure. EKG at that time revealed right axis deviation with sinus tachycardia. There was no response to any therapy.

With reference to the pathological lesions, we do

not feel that we can add much to the already existing detailed descriptions of this ailment.[5] The pancreas can vary greatly in gross appearance from completely normal to a markedly shrunken and distorted organ. Microscopically, the pancreas shows prominent fibrosis of the interstitial tissue with dilated large ducts and smaller ductules (Fig. 2). Eosinophilic staining, viscous appearing material, which may have a concentric appearance, plug many of these ducts (Fig. 3). There is usually a lymphocytic and plasma cell response to the extravasation of this abnormal mucus. Remarkably, the islets of Langerhans are preserved, often showing hyperplasia and lying in close approximation to each other (Fig. 4). We feel

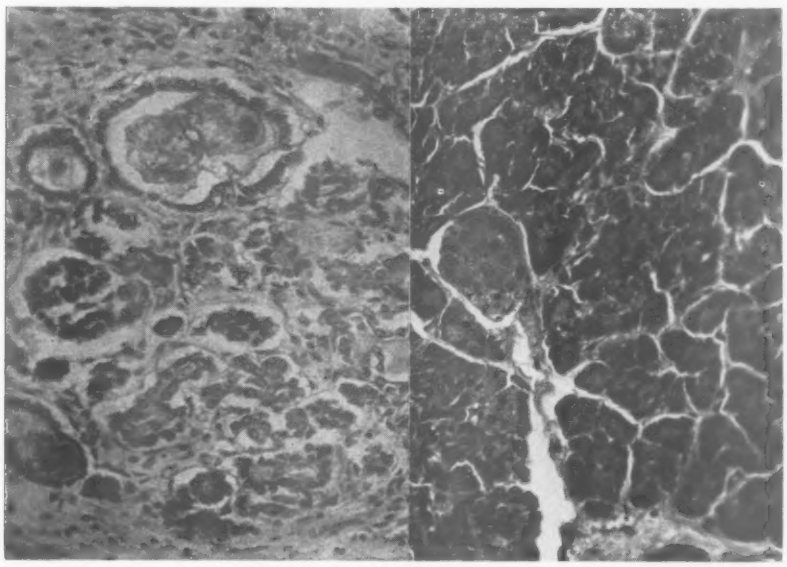

Fig. 2. (A). Pancreas with dilated ducts, atrophy of acini and interstitial fibrosis. Normal pancreas on the right for comparison.

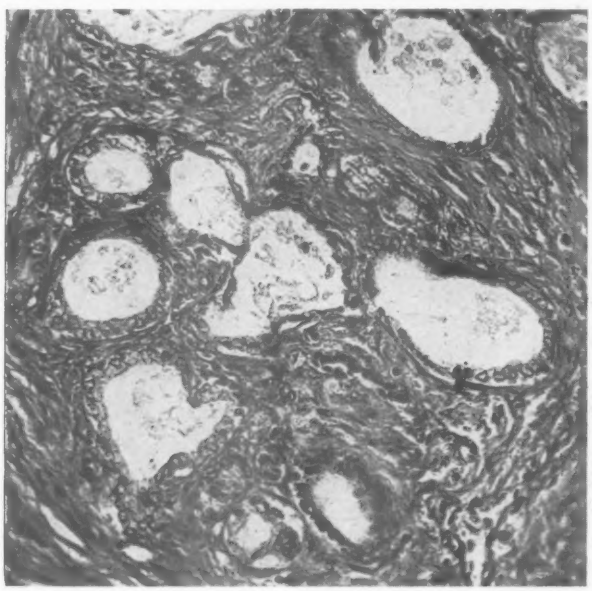

Fig. 2. (B). Cystic pancreatic ducts surrounded by thick fibrous bands.

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that the interesting phenomena of the preservation of the pancreatic islets in this disease is probably best explained as being the result of obstruction of the pancreatic ducts by viscous mucus plugs. Banting and Best,[24] in their original experiments with insulin production, found that after ligation for 7 to 10 weeks of the pancreatic ducts in dogs, the ducts distal to the obstruction were dilated, the acinar tissue

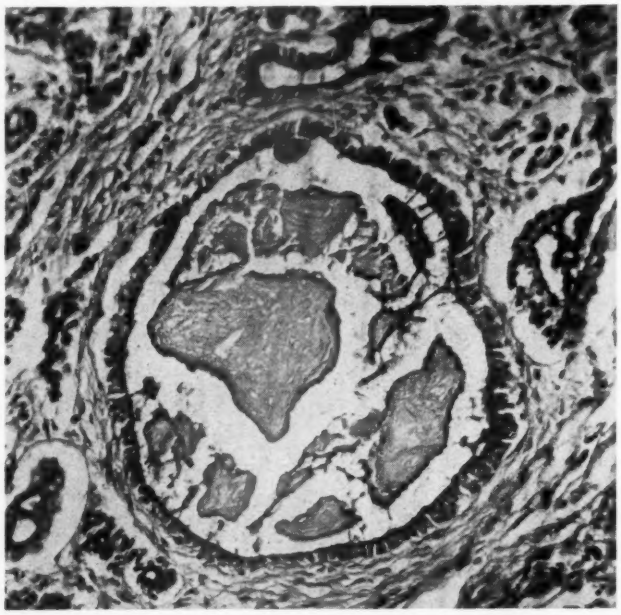

Fig. 3. Eosinophilic, inspissated concretions (mucus secretion) fill a dilated duct.

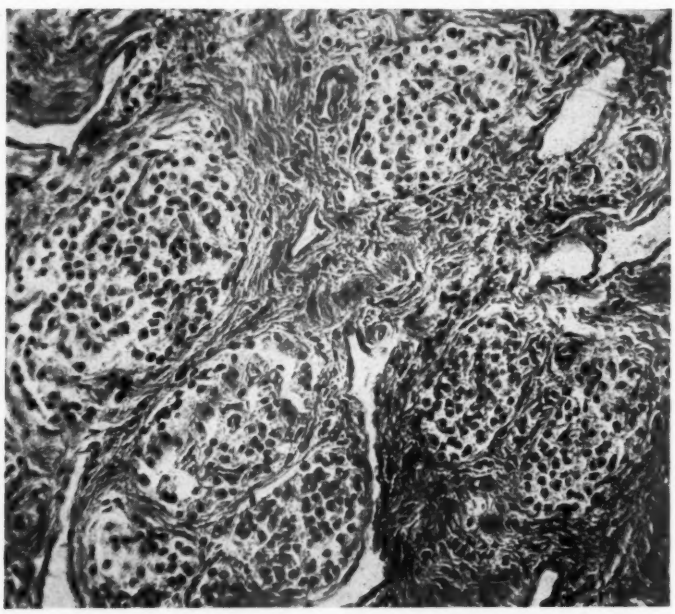

Fig. 4. Islets of Langerhans showing hyperplasia and lying in an adjacent location.

destroyed with fibrous tissue replacement, and the islets were still preserved and remarkably healthy in appearance.

Grossly, the lungs can vary in appearance from normal to that of typical bronchopneumonia and later marked bronchiectasis and abscess formation. The

Fig. 5. Bronchus is plugged with thick mucus, reminiscent of that found in bronchial asthma.

Fig. 6. (A). Lungs are pale and emphysematous with foci of dark atelectasis.

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bronchioles, large and small, may be grossly plugged with mucus (Fig. 5). Microscopically, when the lung is involved, emphysema is present in nearly all areas not replaced by atelectasis, fibrosis, or a bacterial process (Fig. 6). The bronchial epithelium may be edematous and the lumen filled with tenacious mucus. Much of this mucus takes the same eosinophilic stain as already noted in the pancreatic ducts. Irritation results in increasing peri-bronchial and alveolar wall firosis. It is only a question of time unless the patient can be successfully controlled on antibiotics until superimposed infection results. Some feel that the use of the broad spectrum antibiotics may actually cause striking improvement in the general nutritional state.[23] This has not been our experience thus far. The staphylococcus organism has most often been incriminated and the increased flow of abnormal mucus no doubt plays a prominent part in augmenting the invasive characteristics of this organism.[6]

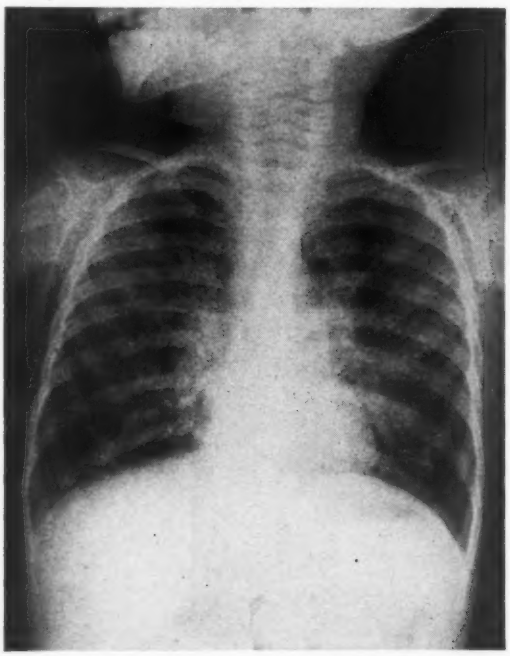

Fig. 6. (B). Diffuse fibrosis throughout lung fields.

Depending upon the degree to which fibrosis has replaced normal lung tissue will the heart itself be involved, resulting in cor pulmonale[9,11] (Fig. 7). This occurred in 17% of 42 cases reported by Nadas *et al.*[22] The thickened right heart musculature with heavy trabeculae in one of our patients with extensive lung disease, is mute testimony to this.

The liver, when involved, shows increase in size with replacement by fat of normal cell structure. The bile ductules may be dilated and filled with mucus

Fig. 7. Extreme dilatation and hypertrophy of the right side of the heart.

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(Fig. 8). Extremely distended and hyperactive glands may be found in the mucosa of the gallbladder.

When severely involved, the mucosal surface of the intestine is plastered with mucus and, microscopically, Brunner's glands in the duodenum are found to be over-active and may produce the same eosinophilic staining type of abnormal mucus.

There is an interesting microscopic finding in one of our cases, which to our knowledge has not yet been described in the literature. In this patient the examination of a lymph node from the pancreatic region revealed macrophages laden with eosinophilic staining mucus, signifying perhaps a body so overburdened with abnormal mucus that the defense mechanisms, to get rid of harmful foreign material, were called into active play (Fig. 9).

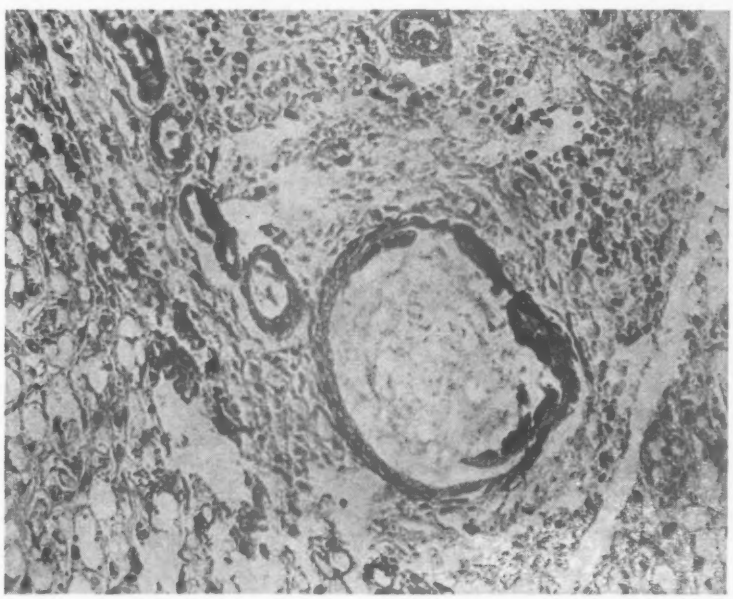

Fig. 8. Bile duct and ductules dilated and contain abundant mucus. Surrounding liver cells are engorged with fat.

If our theory of the pathogenesis of mucoviscidosis is tenable, then the prognosis of this disease should be better than has formerly been thought. Especially should this be true in those individuals whose respiratory tree has been relatively spared. The location, the number, and the extent of involvement of the mucous glands would then directly vary the prognosis. With this in mind, all of the autopsied cases of adults between 1936 and 1951 with the diagnosis of chronic pancreatitis or cystic disease of the pancreas at the Medical College of Virginia Hospitals were reviewed. All those who had any related liver, gallbladder, or gastric disease were discarded, as were those with a history of chronic alcoholism. This left a small group of adults who died of causes apparently unrelated to their pancreas with the same microscopic picture in the pancreas on hematoxylin and eosin stain, as is found in mucoviscidosis in childhood. Two case histories follow:

Case 1. R.J. was an 81 year old, white male, who had suffered with frequent bouts of pneumonia for at least half of his life span, these episodes having been suffiicently severe to require numerous clinic visits and several hospital admissions. The last admission was for cardiac failure which did not respond to attempted digitalization. At autopsy, besides the cardiac findings, the pancreas showed marked peri-acinar fibrosis and cystic dilatation of the ducts and with eosinophilic plugging in numerous areas (identical with Fig. 2B).

Case 2. R.C.G. was a 68 year old, white male, who had undergone a cholecystectomy several years previously for abdominal pain without relief. Two subsequent laparotomies were done without significant findings. (This type of history is frequently obtained in long standing cases of chronic pancreatitis.) Twenty-four hours before suffering a myo-

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cardial infarction and sudden death, he had copious watery stools. Other than the cardiac pathology, autopsy revealed the typical picture of chronic pancreatitis.

Frequently the dilated duct lumina of chronic adult pancreatitis contain eosinophilic staining, often laminated, viscid appearing material which is thought to be a mucoprotein secreted from the remaining acinar cells and the epithelium of the ducts.[10] Microscopic examination shows peri-acinar fibrosis. Pancreatic stones may or may not be a feature.

After all known causes of chronic pancreatitis in adults have been ruled out, there still remains this group in which this characteristic pathology occurs and who may never have suffered any known pancreatic discomfort in life. Recent investigators remark, "Though chronic pancreatitis may become manifest by all the symptoms and signs, it is most puzzling that severe morphologic changes can occur in the organ and the patient remain symptomless."[10] These authors present sixteen such cases and describe the eosinophilic staining, inspissated concretions in the pancreatic ducts which they believe to be abnormal mucoprotein and to form the nidus upon which calcium may be precipitated to form stones. They believe that the quantity, pH, and enzyme content of pancreatic juice varies with the stimulating agent. The juice formed by vagal or pilocarpine stimulation, which is more constant in effect, is smaller in quantity and less alkaline, but is rich in enzymes. Following a meal, a free flow of highly alkaline juice, poor in enzymes, is produced which serves to flush out the ducts, carrying with it the more viscid enzyme rich material.[10] This implies that in a certain group of adults with chronic pancreatitis, excessive vagal stimulation, at the expense of the sympathetic component, may be the basic cause of the disease. These same investigators have found that the normally alkaline pancreatic juice, which is supersaturated with calcium carbonate, can be made more alkaline by excessive mucoprotein secretion, causing calcium to be precipitated, and thus stones to form.[10]

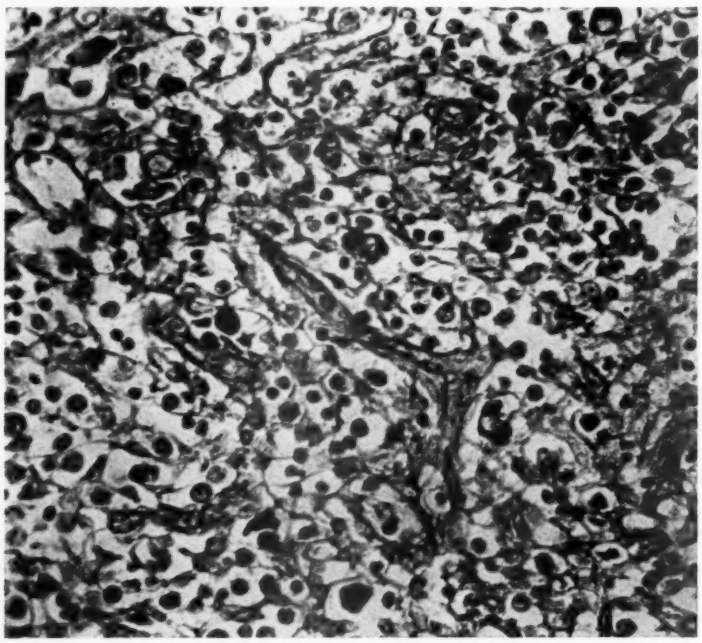

Fig. 9. Dark staining histiocytes filled with mucus. P.A.S. stain.

This, then, reveals that in a certain group of adult patients with chronic pancreatitis, with or without stone formation, the pathogenesis is strikingly similar to that of mucoviscidosis and *may* represent a subclinical form which has held over into adulthood. At least one other case of possible cystic fibrosis of the pancreas, diagnosed by autopsy, in a 34 year old Negro male is on record.[16] It is an intriguing thought that a minimal involvement of

the mucus secreting glands in the original entodermal tube may, in the last analysis, be the cause of a significant number of thus far unexplained cases of chronic pancreatitis in adulthood.

Addendum

Since the completion of this paper we have had the opportunity of studying three additional cases diagnosed pre-mortem and proven by autopsy. Two of these cases were admitted to the Medical College of Virginia and the third was diagnosed by William M. Bruch, M.D., Bluefield, Va., and was autopsied there by one of us (G.R.H.).

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INFLAMMATORY RINGWORM IN MAN DUE TO TRICHOPHYTON FAVIFORME CONTRACTED FROM CATTLE*

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Inflammatory fungous infections of the skin occurring in man and acquired from cattle have been known in Europe for many years. These infections are frequently caused by the faviform trichophyta which were first studied by Sabouraud[1] in 1893.

Recognition of such infections in this country is of more recent date. The first Canadian report in 1934 by Davidson, Gregory and Birt[2] described 28 cases, 9 of which were proven by culture to be caused by *Trichophyton faviforme*. In 1938 Gammel and Work[3] reported three cases, one of which was from the United States. Fowle and Georg[4] described 14 cases from Pennsylvania in 1947 and this was followed by Carney's[5] publication from Iowa describing 39 cases of inflammatory ringworm, 17 of which were caused by the faviform trichophyta.

The increasing number of reports of such infections indicates not only the likelihood of a greater incidence of this type of ringworm in the United States, but also a better understanding of the culture methods necessary for isolation of the faviform trichophyta. To further focus attention on the increasing incidence of this disease, we are reporting five cases of recent occurrence in Virginia. Since the methods for successful cultivation of the faviform trichophyta vary somewhat from the generally used methods for growth of the ringworm fungi, we are reporting them here in some detail.

Fungous infections of the skin due to the faviform trichophyta appear in at least three forms. In the scalp kerion formation is described, identical clinically to that caused by *Microsporum lanosum*. In the bearded region, indurated boggy areas, somewhat kerion-like occur and are confused with infections in this region due to bacteria. On the glabrous skin, the organisms may produce either inflammatory circinate tinea, with or without pustules, and agminate folliculitis. All three of these forms were encountered in our cases.

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CASE REPORTS

Case 1.—J.H.M., a 35 year old male, noticed a few pustules on the left side of the chin about April 3, 1952. Prior to this time he had been treating cattle for ringworm with a preparation supplied by a veterinarian. The disease in the animals was predominantly about the head and appeared as dry grayish scaling plaques which were accompanied by hair loss in the involved areas. Upon examination of the patient on April 10, 1952, there were noted three groups of pustules on the bearded region surrounded by an indurated elevated erythematous plaque which was fairly well defined. The pustules when opened discharged a slightly bloody purulent material. The hairs in the area were easily epilated manually. Up to this time the patient had used a salve without success. Cultures for bacteria and fungi were taken and since we were then unfamiliar with cattle ringworm in this part of the country, the patient was placed on wet compresses of boric acid, neomycin ointment locally and penicillin internally. Four days of this therapy had very little effect upon the lesions. Within the next 10 days he received two fractional doses of 75r x-ray, unfiltered, parenteral penicillin, and, because of the history of exposure to cattle ringworm, was placed on oral potassium iodide beginning with 15 drops t.i.d. On the eleventh day of treatment aureomycin was combined with the penicillin. Two days later a fungus was found to be growing on the Sabouraud media and all antibiotics were discontinued. The patient was treated for a total of six weeks with seven weekly x-ray exposures of 75r unfiltered, wet compresses of boric acid and 8% ammoniated mercury ointment. The potassium iodide was gradually increased to 30 drops t.i.d. A faviform trichophyton was recovered from the culture.

This case illustrates the ineffectiveness and even possible exaggeration of the disease by the use of antibiotics when fungi are not suspected.

Scales were obtained from the infected cattle and were found to contain *Trichophyton faviforme.*

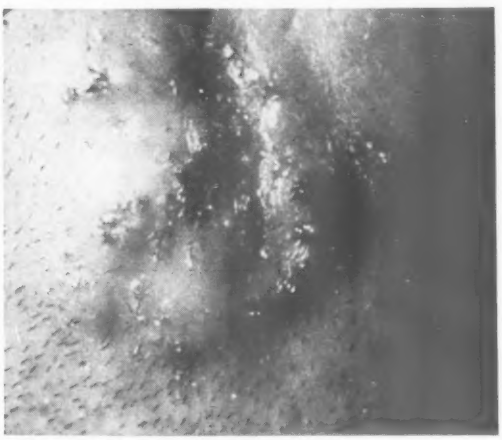

Plate I. Kerion-type lesion of the beard region.

Case 2.—H.D., a 24 year old farmer, was seen in May, 1952, complaining of an eruption on the right side of the neck of one month's duration. He had come in daily contact with cattle but was unaware that any were diseased. There had been considerable local treatment, including wet compresses, to no avail. On examination there was an indurated area 4 x 6 cms. in diameter on the right side of the neck below the mandible. Grouped in this erythematous plaque were multiple pustules from which slightly sanguineous purulent material was expressed. Direct examination of hairs were positive for fungi. The hairs in the area were manually epilated. The patient was placed on potassium iodide internally and boric acid compresses followed by 8 per cent ammoniated mercury ointment three times daily. Over a period of six weeks he was given weekly fractional doses of 75r x-ray, unfiltered, to the involved areas and was clear at the end of this time. Cultures revealed the presence of *T. faviforme.*

Case 3.—W.S.P., a 35 year old business man was examined on August 15, 1952, complaining of an eruption on the left cheek of two weeks duration. He had been treated with wet compresses, local antibictics, ammoniated mercury ointment, and a triple sulfonamide drug internally. During the 10 day period of this treatment he had become progressively worse. There was no history of contact with cattle although he occasionally visited a farm. The lesion on the left cheek measured 4 x 5 cms. and was indurated, somewhat boggy to the touch and deep red in color. On this plaque were pustules from which a moderate amount of pus could be expressed. A direct smear was positive for fungi and *T. faviforme* was later identified from the culture. The patient was placed on potassium iodide beginning with 45 drops and increasing to 90 drops a day. Locally, wet compresses of Epsom salts with 3 per cent iodide crystals in petrolatum were applied three times daily. In addition he was given weekly treatment of 75r unfiltered x-ray, and manual epilation, and was clear at the end of four weeks.

Case 4.—F.R.M., a farmer, age 34, was seen on May 2, 1952, with an annular lesion on the left upper arm, 8 cms. in diameter, of three weeks duration. There was a history of having treated cattle for ringworm. The lesion had an erythematous edge with fine scaling. Throughout the center were multiple papules with vesicles often associated with hair follicles (agminate folliculitis). Direct examination was positive for fungi. The patient was placed on potassium iodide internally and Castellani's fluid t.i.d. externally. At the end of seven days 8% ammoniated mercury ointment was applied locally along with weekly fractional doses of x-ray for four weeks. The patient was clear at the end of six weeks. Cultures revealed *T. faviforme.*

Plate II. Pustular agminate folliculitis occurring in hairy areas.

Case 5.—Mrs. F.R.M., wife of Case 4, had helped her husband treat the cattle. When seen on May 2

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there were lesions on the left shoulder of three weeks duration. The lesions were very superficial, 2 cms. in diameter, with a circinate, erythematous border. The patient was given one exposure of 75r x-ray, placed on potassium iodide internally, and given Castellani's fluid for local use three times daily. While this lesion was thought to be very superficial and clearing was expected early, yet four weeks were required for cure. *T. faviforme* was recovered from the culture.

Plate III. Circinate lesions on glabrous skin with some vesiculation.

There is considerable difference of opinion in regard to the treatment of cattle ringworm in man, although the duration of treatment varies only from four to six weeks. Some authors prefer iodides internally, ammoniated mercury externally, with or without x-rays. Carney[5] stated that his patients, most of which were children, were cured with manual epilation, wet compresses and 3 per cent ammoniated mercury in about four weeks. It is certainly true that some of these infections are self-limiting in view of the beneficial effect of pustulation in helping to bring about evacuation of fungi. However, if the simple treatment as outlined by Carney seems to have little effect, as was evident in three of our cases, we believe iodides and x-rays of considerable value. In our hands, x-ray has seemed to be of benefit in reducing the uncomfortable inflammatory reaction, and is not intended to be fungicidal.

MYCOLOGY

The suppurative type of ringworm infection of man transmitted by cattle is usually caused by one of the faviform trichophytons. There appears to be a difference of opinion among mycologists as to the relationship to one another of the three organisms generally involved — *Trichophyton album, Trichophyton discoides* and *Trichophyton ochraceum.* Conant and his collaborators[6] claim that these so-called species are synonymous with *Trichophyton schonleini,* the causative agent of favus. MacKinnon and Artagaveytia-Allende[7] believe the three may be considered as synonyms but that they are quite distinct on a botanical, chemical, and epidemiological basis from *T. schonleini.* Georg[8] has suggested that they be considered as varieties of a single species—*Trichophyton faviforme.* We are more inclined toward the latter view, although in our experience we have not found the distinguishing characteristics between *T. faviforme album* and *T. faviforme discoides* to be clear-cut.

The infecting agents isolated from the four human cases and also obtained from one of the cows were identified as *Trichophyton faviforme** and according to cultural appearance were classified by us as belonging to the album variety.

Certain unusual features of *Trichophyton faviforme* as seen in direct examination of material from the lesion and also in culture may be confusing to anyone not familiar with this species of dermatophyte, and for this reason we are describing the laboratory observations as an aid to diagnosis.

Direct Examination: The fungous elements may be seen in wet mounts or gram stained smears of material expressed from the pustules present on the surface of the lesion. In the latter preparations, where not too strongly decolorized, the fungus spores usually appear as gram positive, round or oval bodies, 3 to 4 microns in diameter lying singly, in pairs or occasionally in chains of three or more elements among the leucocytes. Rarely short chains of three or more rectangular cells may be seen. The spherical form and relative scarcity of the fungous elements is in contrast to the numerous branching filaments generally seen in the common ringworm infections of man and may be confusing unless the examiner has had previous experience with the faviform trichophytons. The Hotchkiss-McManus stain is also satisfactory for direct examination but shows less contrast than does the gram stain.

Culture. Various authors, including MacKinnon[9], Robbins, MacKinnon and Ma[10], Fowle and Georg[4],

***Confirmation of this identification was made by Dr. Libero Ajello of the Communicable Disease Center, Chamblee, Georgia.**

MacKinnon and Artagaveytia-Allende[7], have studied the vitamin requirements of the faviform trichophytons and pointed out the importance of certain members of the vitamin B complex in their nutrition. Georg[8] has suggested the use of Sabouraud agar supplemented with thiamine or (difco) blood agar base as suitable culture media for isolation and culture of this species. Growth occurs more rapidly at 30°C. to 37°C. than at room temperature.

Our initial isolations grew well on blood agar, but poorly or not at all on Sabouraud dextrose agar. However, when a thiamine supplement of 100 mcg. per cent was added to the latter medium, good growth ensued. In contrast to the majority of dermatophytic fungi of man, growth takes place slowly, individual colonies attaining a diameter of only 4 mm. to 5 mm. after ten days. The colony is glabrous in appearance with a raised, vermiculate center and adheres closely to the medium. As growth continues, the colony becomes much folded with sloping sides upon which a white, downy growth appears. Upon microscopic examination of the colony, irregular, many-branched hyphae may be seen. Terminal and intercalary chlamydospores are numerous in some colonies. Georg[8] has described the appearance of microconidia and macroconidia typical of the Trichophyton genus when these fungi are grown on media supplemented with thiamine.

SUMMARY

Five cases of inflammatory ringworm in man due to *Trichophyton faviforme*, the common agent of ringworm infection of cattle, are presented. The source of infection is clearly indicated by the histories of the patients, four of whom were in daily contact with cattle. In three cases the patients had been treating ringworm infection in these animals. *T. faviforme* was cultured from the skin lesion of one of the animals.

Since laboratory procedures for the successful growth and identification of *T. faviforme* differ somewhat from those used routinely for the majority of dermatophytes, suitable methods are discussed.

A method of therapy is outlined which is of particular value in resistant cases.

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ADVANCED CARDIOSPASM ASSOCIATED WITH EXTREME MALNUTRITION—RELIEF BY DILATION—REPORT OF A CASE

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Failure to relieve cardiospasm by dilation is almost, if not always, the result of inadequate stretching of the cardia. Unless a swallowed silk thread is employed as a guide in introducing dilating instruments into the area of obstruction, relief from dysphagia will not be provided. Even when a guiding thread is utilized a hydrostatic dilator may slip above or below the segment of obstruction while it is being distended and effectual stretching is not obtained. In such instances symptoms are not relieved and frequently patients are advised to have some type of surgical treatment "because dilatation has failed to afford relief from dysphagia."

We have seen many patients with cardiospasm who have not been relieved from symptoms following dilation treatment in whom we have reason to believe that the cardia was really never adequately stretched.

At times during dilation with a hydrostatic dilator the instrument can be felt to pull into the stomach or push back into the esophagus during inflation. When this is noted relocation of the dilator is imperative if satisfactory dilation is to be obtained.

In other cases the dilator may apparently remain stationary but, unless expansion of the instrument produces pain at the cardia, effective stretching is rarely provided. Usually a small amount of bleeding is associated with the dilation.

When ordinary conventional methods of dilation fail to afford relief from dysphagia the dilator should be passed into the stomach and distended. Then the expanded instrument should be pulled forcibly, elevating the cardia as high as possible. Water pressure is then relaxed and after the dilator slips into the esophagus for two or three inches the pressure is rapidly renewed. This procedure always results in adequate dilation and relief from symptoms. This manipulation is associated with additional trauma and should be reserved for patients who are not relieved by ordinary methods of dilation.

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At times patients may have difficulty in swallowing a thread because of marked esophageal obstruction, lack of cooperation, or as the result of unexplained tangling with the formation of knots that prevent free passage of the dilator into the cardia. Perseverance and encouragement will almost always overcome these obstacles.

Infrequently marked starvation and associated mental apathy will present extremely difficult problems as indicated in the patient who is herewith reported. Most of these problems are not insurmountable, as illustrated in this unusual case.

CASE REPORT

The patient—a Negro man—was first examined and treated here at the age of 18 in 1930 for a gunshort wound of the abdomen. A perforation of the duodenum was repaired and the patient made an uneventful recovery. At his second admission in 1942, he complained of difficulty in swallowing that had been present for several years. The symptoms were typical of cardiospasm and roentgenoscopic studies revealed obstruction at the cardia with moderate dilation of the esophagus above the point of narrowing.

Following the passage of a sound, size 60 French, over a guiding thread, the patient was relieved from dysphagia, but was instructed to return for hydrostatic dilation of the cardia in case obstruction to swallowing recurred. Reappearance was deferred until December 18, 1952, when he entered the hospital in a deplorable state of starvation, having lost weight from 120 to 63 pounds (Fig. I).

Apparently relief from dysphagia was temporary, as is frequently the case unless the cardia is stretched with a hydrostatic dilator.

His blood count revealed a hemoglobin content of 3. grams per 100 cc. and the red cell count was 1,400,-000. The patient was apathetic and made no effort to help himself in any way. Roentgenoscopic examination revealed a widely dilated esophagus filled

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with food and secretion (Fig. II). Other studies showed evidence of syphilis of the central nervous system. Blood transfusions, penicillin in large amounts, and vitamin products were introduced in every possible way. As was to be expected all efforts to have the patient swallow a thread were fruitless.

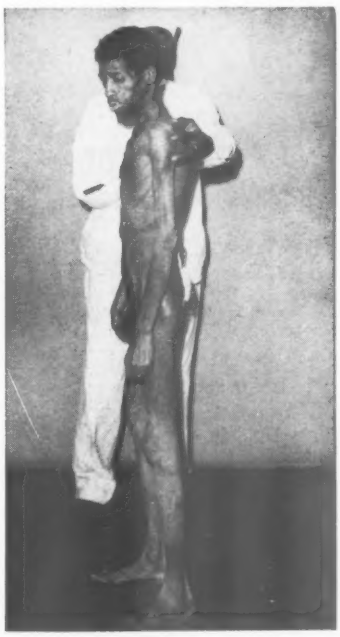

Fig. I.

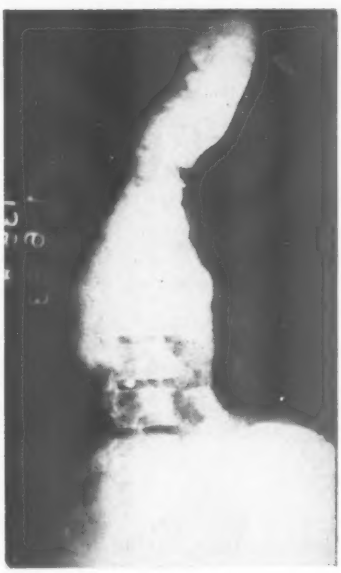

Fig. II.

Supportive treatment was followed by sufficient general improvement to permit esophagoscopy on January 9, 1953. After the esophagus had been thoroughly evacuated of secretion, the cardia was located and the esophagoscope was passed into the stomach. A metal bobbin with thread attached was placed in the stomach. Three days later sufficient thread had passed into the intestinal tract to permit dilation of the cardia with a hydrostatic dilator.

Following treatment the patient was able to swallow normally and was sufficiently improved to return to his home on January 16, 1953.

Fig. III.

At his last examination on April 16, 1953, patient looked well and had not noted any further difficulty in swallowing. He had gained forty pounds in weight. (Fig. III).

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CANCER OF THE THYROID
INCIDENCE AND RELATION TO THYROID DISEASES IN THE TIDEWATER AREA OF VIRGINIA AND NORTH CAROLINA.*

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The ominous sign of a solitary node in the thyroid gland is still not sufficiently appreciated. In spite of a number of recent publications indicating the importance of this lesion, it is often neglected by physician and patient. On the other hand, two recent articles,[15,16] based largely on autopsy material, have tended to minimize the importance of the solitary node. Fortunately, Cope[5] and others[3,12] have questioned the validity of this approach. They pointed out that most patients with cancer of the thyroid die at home and therefore do not come to autopsy. In our experience the physicians of Tidewater likewise disagree on the necessity of removing the solitary node. Accordingly, we felt that it would be of value to study the thyroid lesions of this area. Since pathologic manifestations in this organ show marked geographical variations,[1,2,4,10] it would be of additional interest to examine the distribution of the various diseases of the thyroid in the Tidewater area of Virginia and adjacent North Carolina. To our knowledge, such a study has not been made.

The material consisted of all the surgical specimens collected over a three year period (1947 to 1950) from the pathological laboratories of the DePaul, Leigh Memorial and Community Hospitals of Norfolk and of King's Daughters' and Maryview Hospitals in Portsmouth. Patients from nearby North Carolina as well as from Virginia are treated in these institutions. This material also includes specimens sent in from the adjacent Tidewater area of these two States.

A total of 154 cases was studied. Of these patients, 79 were born in Virginia and North Carolina, and 46 elsewhere. The birthplace of 29 patients was unknown. Table 1 shows the incidence of the various diseases encountered. In the total material there were twelve cases of cancer, giving a percentage of 6.5. No agreement exists on the microscopic diagnosis of malignancy in the thyroid. Therefore, our criteria should be stated. Infiltration beyond the capsule and unquestionable blood vessel invasion (demonstration of the point of invasion in the section or thrombosis) were required for the diagnosis of cancer in debatable cases. Under these criteria none of the tumors with benign cell picture was classed as carcinoma. Table 2 shows the different types of cancer encountered. The most common type was the low grade papillary adenocarcinoma. Attention is called to the fact that the average age of these patients was 31 and three of the six were under the age of 30. Tumors of moderately high grade malignancy, the alveolar carcinomas, had a higher age incidence. This was also true of the single case of high grade anaplastic carcinoma. It is interesting to note that in only one case was a pre-operative diagnosis of cancer made and, in this patient, the lesion was inoperable.

Two of the cases of low grade malignancy belong to the group previously classified as "lateral aberrant

TABLE 1
INCIDENCE OF THYROID DISEASE IN 154 SURGICAL SPECIMENS

Diagnosis	No.	Per cent	Sex M	Sex F	Race W	Race C	Avg. age	Age range
Diffuse non-toxic	2	1.3	1	1	1	1	48	37-58
Nodular non-toxic goiter	50	32.5	1	49	34	16	43	16-67
Toxic Goiter	47	30.5	5	42	31	16	41	19-68
Diffuse	32		3	29	18	14	36	19-55
Nodular	15		2	13	12	3	53	44-68
Adenoma*	37	24.0	5	32	31	6	42	24-72
Carcinoma	10	6.5	2	8	9	1	42	24-68
Thyroiditis	8	5.2		8	8		47	23-64
Lymphomatous	5			5	5		44	23-58
Subacute Giant Cell	1			1	1		49	
Fibrosing (Riedel)	1			2	2		60	55-64
Total	154	100%	14	140	114	40		

*Colloid 23; Microfollicular 6; Fetal (tubular and trabecular) 6; Papillary 2.

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*Read before the annual meeting of The Medical Society of Virginia, at Virginia Beach, Va., October 7-10, 1951.

TABLE 2
CANCER OF THYROID—10 CASES

Type	No. of cases	Avg. age	Age range	Sex male	Sex female	Clinical Diagnosis
Papillary	5	31	24-42	2	3	2 Lymphadenopathy 3 Benign Adenoma
Alveolar	4	50	37-65	-	4	4 Benign adenoma or nodular goiter
Anaplastic	1	68			1	Carcinoma

thyroid." Until a few years ago it was widely believed that this benign appearing thyroid tissue occurring in the lateral part of the neck was a congenital abnormality. Recently it has been shown that it is a true metastatic growth and that the primary tumor can always be found in the thyroid gland. It is generally small, sometimes so minute that it can only be found microscopically.[6,8,11,13,19] In the two patients mentioned a lymphnode lateral to the carotid sheath was excised and was found to contain thyroid tissue. According to the modern concept of these tumors, the adjacent thyroid lobe was later removed and, in each case, the primary carcinoma was discovered. These primary tumors were characteristically small, one measuring 5 mm. and the other 2 mm. in diameter.

No cases of cancer were found in association with diffuse toxic goiter or with nodular goiter. The rarity of malignancy in toxic goiter is well known[14].

We were particularly interested in establishing the relationship of carcinoma to the solitary node. Forty-two of our 154 cases were clinically diagnosed as benign solitary adenoma. Five (12%) of these proved to have carcinoma.

The number of our cases is obviously too small to permit precise statistical conclusions. However, various points valuable for discussion are clearly shown. There is no "cancer age" for the thyroid. One-half of the cases of cancer were under the age of 40, and a third were under the age of 30. There were no children in this group, but the great danger of a solitary node in the thyroid of a child has been emphasized by others. In children under 13 years of age 55% of solitary nodules are malignant[2]. The younger the patient the greater is the danger of malignancy[17]. Cancer of thyroid in children develops most often at the age of early puberty; it also can occur in the pre-school age[9].

The single node is the point of attack in the prevention and cure of thyroid cancer. The incidence of malignancy in these lesions is amazingly high. As previously mentioned it was 12% in our group. Lahey and Hare[12] in reviewing a large series of cases report carcinoma in 10.04% of the solitary nodules. Others[1,2,4,5,18] have found the incidence of malignancy to be from 9 to 24%. G. Ward and his co-workers[17] believe that approximately 80% of malignancies originate in discrete adenomas and that 5 to 20% of discrete adenomas will eventually become malignant, a point which is difficult to prove.

There is general agreement that by clinical examination it is impossible to tell whether or not a node is malignant. Waiting for a definite diagnosis means waiting for inoperability. Anglem and Bradford[1] point to the folly of making the diagnosis by observation of continued growth until the malignant nature is clear and unmistakable. R. Ward[18] states that when carcinoma of the thyroid was diagnosed or suspected, 80% of the patients were dead after 5 years; when the diagnosis was established at operation, 60% were dead after five years; but when the diagnosis was made by pathological examination alone, only 20% had died at the end of five years. All clinically diagnosed cases proved to be of high grade malignancy in Coffey's series[2]. In our group the one cancer suspected pre-operatively was inoperable and highly malignant.

The present approach to cancer is prevention if this can be accomplished without undue risk. The removal of the single node in the thyroid is a harmless operation. Anglem and Bradford[1] report 331 prophylactic operations for a solitary nodule without a death. Lahey and Hare[12] also stress that there should be no mortality or morbidity.

We believe that for the purpose of statistics, as well as for the approach to the patient, a distinction should be made between nodular goiter and the solitary nodule. In Lahey and Hare's[12] series, there was a 10.04% cancer incidence in solitary nodules. However, in patients who had multiple nodes, the occurrence of malignancy was only .62%. Those who group the two entities together get a much higher percentage of malignancy for nodular goiter. It is in the non-goitrous area where the importance of the differentiation between the two can be shown clearly. When we approach the center of an area of endemic goiter where most adults have a nodular mass in the neck, the search for and the evaluation of a single node becomes more and more meaningless

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even though the incidence of cancer rises remarkably.

Small is the number of cancer types which can be prevented. One is carcinoma of the thyroid. Lay propaganda informing the public of the dangerous single node in the thyroid, especially in non-goitrous regions, is necessary to accomplish this aim. A great responsibility rests upon the family physician. It is he who sees the patient first; it is he who by action or inaction decides the final outcome. The argument for prophylactic removal of the solitary nodule is forcefully stated by Lahey[12]: "It can, in fact, almost be said that if every discrete adenoma of the thyroid could be removed in its earlier stages, we would be able to eliminate, almost entirely, the occurrence of carcinoma of the thyroid."

A pre-operative diagnosis is generally impossible in curable cases. However, there is a valuable aid in establishing cancer at the operating table. In all of our cases the diagnosis of malignancy was made by the fresh frozen section with the exception of the lateral aberrant cancers in which this examination had not been requested. However, this method is limited in its value in that there is usually not sufficient time to make a careful search for invasion of the capsule and blood vessels. The rapid frozen diagnosis is of special value in a diffusely enlarged thyroid. It distinguishes readily between a fibrosing carcinoma and the wooden goiter of Riedel. It recognizes the lymphomatous goiter of Hashimoto and the subacute giant cell thyroiditis. In these two diseases, the value of thyroidectomy is questionable. It is, therefore, important that they not be treated as malignancy. X-ray therapy is desirable in subacute thyroiditis. In struma lymphomatosa x-ray or a conservative resection is indicated because it prevents the patient from developing hypothyroidism or myxedema, such as is commonly observed after extensive surgical removal of the gland in this condition.

The diagnostic value of the Delphian node which lies just above the middle of the thyroid over the cricoid cartilage was recently pointed out by Cope and his co-workers[5]. They state that this node can often be felt pre-operatively because there is no overlying platysma muscle in this region. In the thick lateral part of the neck small metastases are not easily felt. At operation the Delphian node should be exposed as the first step and removed for a fresh frozen section. If this node is positive for cancer, cutting into tumor tissue for diagnosis is eliminated. In 13 of Cope's cases of carcinoma, the Delphian node was invaded 9 times, and it was palpable pre-operatively 5 times. Though no systematic search was made for the Delphian node in our group of cases the presence of metastatic tumor in this node established the diagnosis of cancer in one of these patients.

In conclusion, only one point will be emphasized. In this non-goitrous area, the incidence of thyroid carcinoma is low, but it stands high on the list of preventable cancers. The single node in the thyroid must be removed.

SUMMARY

The incidence of the various surgical diseases of the thyroid in the Tidewater area is tabulated and reviewed.

The incidence of cancer in solitary nodes was 12%.

The danger of a single node in the thyroid is stressed, and its prophylactic removal is urged. Lay propaganda is recommended.

The Delphian node and the value of the fresh frozen section are discussed.

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AN APPRAISAL OF ANTICOAGULANT THERAPY OF MYOCARDIAL INFARCTION*

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The anticoagulants were first introduced for treatment of myocardial infarction in 1945[1]. Since then a voluminous literature concerning their use has appeared. However, despite the passage of eight years, there is no unanimity of opinion concerning their value. Wright[2] considers that their efficacy has been proven, and states they should be administered in every case of myocardial infarction. Russek[3], after extensive investigation, believes that they should be used in only "poor risk" patients. Others[4,5] have cast doubt on their worth in this disease. Widespread publicity, both lay and medical, has been focussed upon the subject. Many thoughtful physicians, who are yet unconvinced or uncertain of their value, have expressed concern about possible litigation for malpractice if a patient under their care with myocardial infarction were to succumb without having received anticoagulants[3]. It is the purpose of this paper to review the pertinent recent literature and draw some conclusions applicable for the average practitioner of medicine confronted with this problem.

Wright and his associates[6], in the American Heart Association study of a series of 1031 cases, reported that the mortality rate in myocardial infarction could be reduced from 23.4 per cent to 16 per cent. This is a statistically significant figure for such a large series, and would superficially seem to have proven the case for anticoagulants. However, certain deficiencies of this study have been pointed out[4]. Supposedly only patients admitted on odd days received anticoagulants, and those admitted on even days none. In the final study there were sixty-three more patients in the treated group than in the control, which could not have happened by chance[4]. Cases were admittedly shifted from one group to the other. Twelve per cent of the control group received anticoagulants at one time or another. It is obvious that strict random selection of patients was not carried out and the statistics based on this study lose much of their meaning. Many believe that this study cannot be used for the final evaluation of the subject because of these discrepancies.

Russek[3] has demonstrated that cases of myocardial infarction can be successfully divided into "good" and "poor risk" groups on the basis of information available on the day of admission. "Poor risk" patients were selected on the basis of severe shock, intractable pain, history of previous myocardial infarction, cardiac enlargement, gallop rhythm, arrhythmias, heart failure, and complicating diseases. He states that the prognosis of "poor risk" patients can be improved with anticoagulants. "Good risk" cases, which make up about forty per cent of his large series, have a mortality rate in those surviving the first forty-eight hours of 1.2 per cent, which could not be appreciably affected by this therapy. Therefore, he concluded that "good risk" patients have little to gain from this therapy as compared to the danger of its use. Furman *et al.*[7] have recently further divided their patients into three prognostic categories of "good", "poor" and "very poor risks". They found that the "good" and "very poor risk" patients were not benefited, but that mortality was lowered in "poor risk" patients.

Bresnick,[8] in a well studied series at the Boston City Hospital, found no essential difference between control and treated groups. Similar results were published in another study[9]. Russek[10] in 1953 found that about one-half of a large group of specialists that he polled did not use anticoagulants routinely in the treatment of myocardial infarction.

The question of the value of anticoagulants in preventing fatal pulmonary embolization from thromboembolic disease in general has been raised. In two large hospitals[11,12] deaths due to pulmonary emboli have risen in recent years, despite the widespread use of anticoagulants and vein interruption.

The danger inherent in the administration of anticoagulants has not received due emphasis. The reported deaths from dicumarol now exceed one hundred[3], and undoubtedly these are only a fraction of the actual number. Many physicians on conversation will mention cases they have personally encoun-

*Presented at the Opening of the New Wing, Clinch Valley Clinic Hospital, Richlands, Va., July 8, 1953.

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tered of deaths due to hemorrhage from dicumarol that have not been reported. Major hemorrhage has occurred in approximately 2% of patients treated with anticoagulants[13].

The initial dose of dicumarol is from 200 to 300 mgms., and the maintenance dose is usually from 50 to 100 mgms. a day. Most authorities believe the prothrombin concentration should be kept between 20 to 30 per cent of normal. There is a wide individual variation in response. From personal experience in the treatment of hundreds of cases of all forms of thrombo-embolic diseases at the Johns Hopkins Hospital it was found that the maintenance dose varied from as much as 25 to 200 mgms. per day. The dose for each individual can only be determined by trial. Daily prothrombin times are necessary for control of dicumarol therapy. It is well to remember that the effect of any one dose is usually apparent 48 hours later, and this should be kept in mind in ordering the drug. Prothrombin times are difficult to perform by the inexperienced, and only technicians trained in their performance should be employed. Most laboratories employ some modification of the Link-Shapiro one stage method. It is also necessary to know what thromboplastin is used, as Conley[14] found that different commercial thromboplastins gave different prothrombin dilution curves. The therapist should know the thromboplastin and method used in his hospital, and the dilution curve for that thromboplastin and method.

Over a period of four years from 1948 to 1952 at the Johns Hopkins Hospital the author gained personal experience in the administration of dicumarol to several thousand patients. During that period of time there were no known fatalities due to anticoagulants at that hospital. The system in use there could well be adopted by others. The prothrombin times were performed in a central laboratory and the dicumarol therapy was ordered by the director of that laboratory and his assistants. Only one person in the hospital ordered the drug at a time. Thus only a few individuals with wide experience administered this therapy instead of many.

Other anticoagulants with similar properties as dicumarol have been introduced. Tromexan and phenylindanedione (Hedulin) have received the most study. On the whole, the reduction of prothrombin concentration with these drugs appears and disappears more rapidly[2]. It is more difficult to maintain a stable prothrombin level because of these effects, and most authorities feel that dicumarol is the most satisfactory drug of this type presently available. The therapist should choose one of these agents and master its use. For maximum anticoagulant benefit for the first forty-eight hours, heparin should be used until dicumarol becomes effective.

Vitamin K_1 intravenously in 100 mgm. doses is an effective antidote for over-dosage of dicumarol, tromexan, or phenylindanedione. The prothrombin time is usually lowered to safe levels in four hours. This material is far more effective than Hykinone (vitamin K), in 72 mgm. doses, which was previously employed, and should replace it for general use. It should always be available whenever and wherever dicumarol or related drugs are being administered.

The recognized contraindications for dicumarol therapy are as follows[2]: hypoprothrombinenia due to vitamin K deficiency or liver disease, subacute bacterial endocarditis, renal insufficiency, blood dyscrasias with abnormal clotting mechanisms, accidental or surgical trauma to the brain or spinal cord, vitamin C deficiency until corrected, benign or malignant ulcers of the gastor-intestinal or genito-urinary tract.

SUMMARY

Anticoagulant therapy is not indicated routinely for the treatment of myocardial infarction as "good risk" patients have no demonstrable need for it. "Poor risk" patients should receive these drugs according to our present knowledge, although the final proof of their value is lacking. Dicumarol should never be given in the presence of recognized contraindications, in the absence of adequate control facilities, or by inexperienced therapists who are ignorant of its proper administration or control. Vitamin K_1 is an effective antidote and should always be available whenever dicumarol is used.

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Describe New Method of Suture Removal.

A new method for the quick, relatively painless removal of surgical skin sutures, by means of a "suture lift," was described in the July 25 J.A.M.A. by Drs. Mandel Weinstein and Morton Roberts, Jackson Heights, N.Y.

The procedure is a very simple one. After the regular stitches have been made, a separate piece of nonabsorbable surgical suture is passed around the loop of the first stitch of the regular suture and tied. The thread is then passed under the loop of the second stitch and around the loop of the third. This procedure is repeated so that every other stitch is fixed to the "suture lift" by an extra turn of the thread.

When the regular stitches are to be taken out, traction upward on one end of the "suture lift" permits the fine thread of the regular stitches to be easily raised, cut and removed, the doctors wrote.

Removal of skin sutures is sometimes very painful because, during the normal healing processes of operative wounds, swelling may cause the suture to be so depressed and buried in the skin that delivering it for severance may be almost impossible, the authors stated. This is especially true if the incision is between skin folds or crease lines.

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RUPTURED LIVER DUE TO PERIARTERITIS NODOSA— REPORT OF A CASE

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Pulaski, Virginia

Periarteritis nodosa, a necrotizing inflammatory lesion of small arteries, arterioles, occasionally also of veins[1], used to be considered a rare disease. Greater awareness of the lesion has resulted in more frequent clinical diagnosis, even though its true incidence may not have increased proportionately. Rupture of the liver as a complication of periarteritis nodosa was reported in 1917 by Klotz[2]. Another case of fatal rupture was reported in 1943[3]. The present case is being reported because of its rarity and in order to illustrate the difficulty of recognizing the causes of acute pain in the abdomen.

CASE REPORT

A 47 year old white male was re-admitted on 9-3-48 complaining of dyspnea, orthopnea, and pedal edema of six weeks' duration. He had been discharged five months previously with a diagnosis of hypertensive arteriosclerotic heart disease, arteriolonephrosclerosis, schizophrenia and calcification of the right renal artery, of unknown etiology. He did fairly well until August of 1948 when the above symptoms reappeared. Initial examination revealed a chronically ill white male with a temperature of 99.8, pulse 120 and blood pressure 244/150. Moist rales were heard at the bases posteriorly. The heart was enlarged to the left, and had a gallop rhythm. Tenderness was noted at both costo-vertebral angles and the liver edge was palpable one finger-breadth below the costal margin. It was tender. Moderate pitting edema of the ankles was recorded.

Laboratory Data: The urine contained albumin (four plus) and a few white blood cells. The erythrocyte count was 4,100,000 per cu. mm. and the hemoglobin was recorded as 13 grams per 100 ml. The Kahn test on the blood was negative. The leucocyte count was 8,900 per cu. mm. with a normal differential; the sedimentation rate (Westergren) was 98 mm. per hour. The non-protein nitrogen was 42.2 mgm. per 100 ml. and the cephalin flocculation was 2 plus/2 plus.

Course: The patient responded well to oxygen, digitalis, diuretics and progressed satisfactorily until his twenty-first hospital day. Then he developed severe pain in his chest and abdomen and his erythrocyte count dropped to 2,300,000 per cu. mm. Two days later it was recorded as 1,500,000 per cu. mm. His blood pressure was 96/56 and he died two days later. The clinical diagnosis was in doubt; dissecting aneurysm, pancreatitis and mesenteric thrombosis being considered.

AUTOPSY

Post-mortem examination disclosed the following pertinent changes. There was an estimated 600 cc. of partially clotted blood in the peritoneal cavity, evidently due to a 6 cm. jagged tear in the right lobe of the liver, which was surrounded by a sub-capsular hemorrhage (Figure 1). The heart weighed 800 grams and the left ventricle was hypertrophied in concentric manner. There was a recent posterior infarction of the heart and arteriolonephrosclerosis and arteriosclerosis, generalized, were found. Adhe-

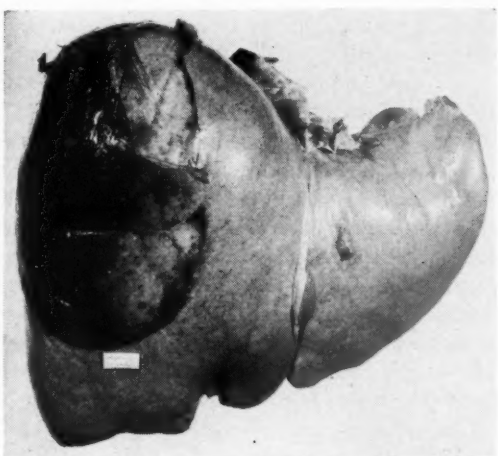

Fig. 1. Linear laceration of liver and extensive subcapsular hemorrhage.

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Reviewed at the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the author are the results of his own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

sive pleuritis was seen on the right. There was aneurysmal dilatation and calcification of the right renal artery. The branch supplying the upper pole of the kidney was almost completely occluded[4].

MICROSCOPY

Infarcts, associated with inflammatory vascular lesions, were found in the heart, testis, brain, gall bladder and liver. The vascular lesions varied in age. In some, there were fibrinoid changes of the vessel wall with thrombosis of the lumen, while others showed "healed" lesions of periarteritis nodosa (Figures 2 and 3).

Fig. 2. Photomicrograph of the gall bladder. In the walls are several vessels with varying stages of vasculitis including fibrinoid necrosis, perivascular inflammation and thrombotic occlusion.

FINAL DIAGNOSIS

Hemoperitoneum due to spontaneous fracture of liver due to diffuse vasculitis (periarteritis nodosa).

Death was considered due to shock and vascular collapse, secondary to the changes listed above.

SUMMARY

A case of spontaneous fracture of the liver with fatal hemorrhage due to periarteritis nodosa is reported. Attention is drawn to the difficulty of diagnosing the etiology of acute abdominal pain.

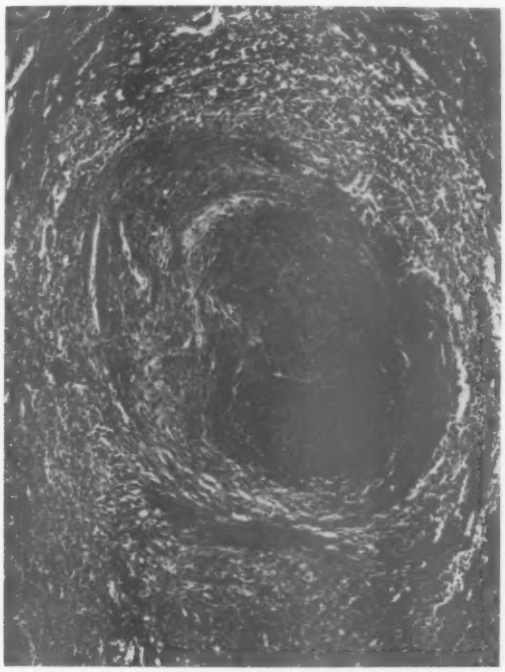

Fig. 3. Higher magnification of one of the vessels illustrated in Figure Two. The vessel contains an organizing thrombus and it is surrounded by inflammatory cells. The walls are sclerotic.

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MEDICO-LEGAL NOTES

Privileged Communications

Physicians are required by professional ethics to preserve the secrets, relating to the performance of their professional duties, communicated to them by their patients regardless of whether these facts are obtained from examination or communications. The extent to which this ethical requirement coincides with the law varies considerably.

In a strict sense the term "privileged communication" applies only to those communications between a patient and his physician, concerning which the patient has a right to prevent disclosure by the physician, in court.

At common law, this type of privilege existed concerning communications between hubsand and wife, and between attorney and client, but not regarding communications between a physician and his patient. The State of Virginia has not passed a statute providing for such a privileged status for communications between patient and physician, and in the absence of such a statute, the courts, during the course of a legal proceeding, may require a physician to divulge anything which has been told to him by a patient, regardless of the objections of the patient. More than a dozen of the states, like Virginia, have never provided for such a privilege. This is not necessarily bad. These statutes have, upon occasion, obviously thwarted justice.

Even in the states which provide, by statute, for privileged communications between patient and physician; evidence obtained from an autopsy is generally held not to be privileged; physician's records contained in hospital records may or may not be privileged, depending on the circumstances; and communications between patient and nurse are generally not privileged. Even though a professional relationship exists, only the information necessary to enable the doctor to act in his professional capacity is privileged. Matters which are entirely distinct from medical facts may be disclosed.

The privilege belongs to the patient and not to the physician. Hence, the patient cannot be forced to testify about his medical consultation anymore than the doctor. Conversely, if the patient consents to the disclosure, the doctor may no longer insist on remaining silent. If the patient sues the physician for malpractice, he immediately waives his privilege.

A second type of communication to which the term "privileged communication" is frequently applied, is illustrated by that type of communication between the physician and a public health authority involving a report of a contagious or a venereal disease, pursuant to the statutory law of the state. In this instance, the physician has a privilege to disclose the information obtained through the medium of his confidential relationship with the patient, in addition to a statutory responsibility to do so. The physician here accrues no liability to the patient for any damage that may ensue, such as the loss of employment or social embarrassment.

The law in many states has been solicitous of the doctor's duty to keep silent on the witness stand, but it has done little to protect the patient's medical secrets from disclosure to other persons in general, except when disclosure of these secrets involves libel, slander, or gross invasion of privacy. The law in this latter area is not well settled due to the absence of statutes, a minimum of litigation, and the necessity of showing damages in order to collect a worthwhile judgment, although the moral or ethical obligation is clear.

*Contributed by Charles W. Whitmore, M.D., L.L.B., University of Virginia Hospital and Medical School, Charlottesville, Virginia.

PUBLIC HEALTH

Mack I. Shanholtz, M.D.
State Health Commissioner of Virginia

Rabies in Man.

The recent death from rabies, August 1953, reminds us of the continued presence of this disease, that it is invariably fatal and that we should carry on our efforts to wipe it out in the animal reservoir and thereby prevent the infection of human beings.

Rabies has been known since ancient times and its symptomatology is characteristic. A history of exposure to infection and the site of infection can usually be obtained. The incubation period may be short, as few as ten days, or long, up to eight or nine months, but usually ranges between fifteen days and five months. During the first two to four days of the onset there may be malaise, headache, elevation of temperature from one to three degrees without fluctuation, anorexia, nausea and sore throat. When there is headache, it is most often localized in the occipital region or over the vertex. There is frequently some abnormal sensation about the site of infection. Bright lights and loud noises may cause convulsive seizures. The onset of the excitement stage is gradual and it is predominant up to death. The most prominent clinical feature is related to the act of swallowing; fluid coming in contact with the fauces is expelled with violence and there are painful, spasmodic contractions of the muscles used in swallowing and of the accessory muscles of respiration. When a patient is known to have been bitten by a rabid animal and when the symptomatology is characteristic, there is little difficulty in making a correct diagnosis.

In the case of the man who died recently, the disease was not suspected at first. There were no open wounds. Respiratory symptoms suggested asthma, from which he had previously suffered. Excitability developed and this, together with the history that the man had, in addition to his full-time job as an automobile mechanic, a part-time job in an animal hospital cleaning cages and holding animals during treatment, led to the consideration of rabies. Death followed within three days and, at autopsy, the diagnosis of rabies was confirmed.

If rabies is to be eradicated from an area, there must be coordination of effort so that control measures will be carried out at the same time in the localities reporting infection. This applies in county, state or country. It requires centralized authority to bring about uniformity of measures. Rabies has been eliminated from several counties in Virginia.

The disease is maintained solely in animals and primarily in dogs. It is usually the local veterinarian who first learns of its presence and he is the person whose cooperation is most desired. Regulations are essential. They include the licensing of dogs and taxation of their owners, the seizure and destruction of all stray dogs, the quarantine or muzzling of all owned dogs while rabies prevails and for at least six months after the last reported case, and the quarantine of all dogs imported into the area for six months. If rabies be prevalent in a community, any dog that has bitten a person should be confined to permit observation for 7 to 14 days and to insure that the animal does not escape if it has rabies. When a rabid dog is found, untiring effort should be made to locate all other animals that were exposed to it so that they may be quarantined.

Vaccination of dogs is recommended; in order to be highly effective it must be combined with other control regulations. To secure prompt recession of rabies and to simplify and aid the collection of stray dogs, a 90 day quarantine for all dogs is advisable.

Rabies in wild animals may be combated by hunting and trapping. The disease cannot persist if the vectors and hosts are drastically reduced among the wild animals in and around a focus of rabies. Without these measures, the disease will kill most of the animals anyway, but the infection will have spread to other areas.

Human deaths from rabies in the past ten years have occurred as follows:

1943—1	1948—0
1944—1	1949—0
1945—1	1950—0
1946—0	1951—0
1947—2	1952—0

Monthly Report of the Bureau of Communicable Disease Control

	August 1953	August 1952	Jan.-Aug. 1953	Jan.-Aug. 1952
Brucellosis	10	6	38	27
Diphtheria	3	4	56	56
Hepatitis	209	64	1642	483
Measles	144	176	4563	15390
Meningitis (Meningococcic)	7	10	144	146
Poliomyelitis	267	268	459	342
Rocky Mt. Spotted Fever	11	18	49	62
Scarlet Fever	19	14	644	547
Tularemia	4	0	22	34
Typhoid & Paratyphoid	11	23	49	68
Rabies in Animals	30	20	328	347
Rabies in Man	1	0	1	0

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MENTAL HEALTH

JOSEPH E. BARRETT, M.D.,
Commissioner, Department Mental Hygiene and Hospitals

Jackie Goes to the Clinic*

Jackie, a twelve year old boy in the fifth grade, is one of many such children in our public schools. Jackie repeated the first and fourth grades, and according to his teacher and his mother, was making little progress in the fifth grade at the middle of the year. It was at this time that Jackie was referred to the Mountain Empire Guidance Center by the visiting teacher at the request of the teacher. The teacher had recognized extreme shyness and a lack of interest in the boy and she was eager to get some help that would result in better school adjustment.

On the report that she sent to the Clinic she stated that Jackie had a history of working and playing alone. While other children were busy with their school activities, Jackie sat and looked out of the window. On the playground he wandered aimlessly around or stood and daydreamed unless the teacher gave him her constant attention.

He lives in a rural section approximately two miles from the nearest home where his maternal grandmother lives.

Jackie's mother and the visiting teacher accompanied him to the Clinic. He appeared shy and retiring but was cordial. The mother said that she was glad to find some help for her son. The history that she gave revealed that Jackie was the youngest of five children. The father had been killed in an automobile accident ten years before while intoxicated, and the mother had worked in a mill in a nearby town since her husband's death. Jackie's older brothers and sisters cared for him in the summer while the mother worked, and during the winter, the maternal grandmother stayed with Jackie during the day while the other children were in school.

Jackie was the only member of the siblings who had some speech difficulty when he entered school at the age of six. He substituted some sounds and omitted several others. At first the family thought his speech was "cute" and would often ask him to say certain things before guests. Later they began teasing him about his speech. The first grade teacher said that she was unable to understand him and recommended repeating the grade.

The mother took Jackie to the family doctor who said that he would outgrow his speech difficulties and that there was no need to be alarmed. But Jackie continued to be more shy and withdrawn, until at the time he entered the Clinic, he was talking very little with anyone. He participated little in class discussions although some of his written work showed evidence of understanding.

When Jackie arrived at the Clinic, he was taken to the playroom and told that he would probably find some books, toys, or games that he might enjoy. At first he looked casually at objects in the room, but later became interested in a phonetic chart with pictures representing the sounds of the alphabet. He asked the therapist about the chart and a phonetic game began. His interest increased as the game continued and when the letter "J" was sounded, he was asked to say his own name, to which he replied, "Ackie". With some help he soon made the "J" sound and used it in front of his name. He then turned to the therapist and said, "That is the first time I have ever said my real name". After that he became more animated and talked at length, stating that no one ever understood him nor liked him at school. When he was told that he could learn to say words as other children, he appeared very pleased and asked to continue playing the "sound" game.

When a psychological examination was administered, Jackie was found to have average ability. His first psychiatric interview revealed anxiety, largely due to his feeling that he was not accepted by his peers.

The Clinic team set to work to reeducate Jackie's family and to help the school understand some of Jackie's problems. An effort was made to help the family sense the importance of making him feel more secure. They were encouraged to give him some important tasks in the home and to listen with interest to his conversation.

Visits were made to Jackie's school where many conferences were held with his teacher and principal. Both were cooperative and the teacher asked for suggestions to help Jackie overcome his shyness and

*Article prepared by Mae Kelly, Psychologist, Mountain Empire Guidance Center, Radford College, Radford, Virginia.

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seeming lack of interest in his school activities. It was suggested that Jackie be treated in the same way as other children in his school activities, thereby giving his peers an opportunity to note his effort and progress and to accept him. Jackie showed rather outstanding artistic ability and it was suggested to the teacher that his productions be used for a school program where posters or pictures were needed.

In the meantime, Jackie was receiving daily speech therapy at the Clinic and meeting regularly with the Clinic team.

At the end of a year Jackie had overcome his articulatory defects to the place that he would make corrections, whenever he made errors, in his conversation. He was participating with satisfaction in his school activities, his Sunday School class, and in the Boy Scouts. His sixth grade work was better than average, and most of all, he appeared to be a happy, well-adjusted boy in his family and in his community.

BOOKS

The Psychology and Psychotherapy of Otto Rank. An Historical and Comparative Introduction. By FAY B. KARPF, Ph.D. Philosophical Library, New York. 1953. x-129 pages. Cloth. Price $3.00.

This book gives more than the title promises. It is not only a comprehensive and intelligible presentation of the psychological views and psychotherapeutical method of one of Freud's earliest associates, but at the same time, an outline of the essentials of psychoanalysis in its original setting and of the systems of two other offsprings of psychoanalysis, Adler's individual psychology and Jung's doctrine emphasizing the synthetical rather than the analytical aspect of psychotherapy. Rank's own thought stands against the background of psychoanalysis as voluntaristic and humanistic against deterministic and mechanistic. Less emphasis is laid by him on the interpretation of the patient's experiences and past than on the "dynamics" of the relationship in the interview. More room is left for values, "spiritual immortality as against sexual mortality" (p.109). In brief, in Rank's teachings, ego and consciousness emerge as factors of greater importance for human life and recovery from neurosis than instincts and impulses.

W.R.

New Books.

Some of the newer books in the Tompkins-McCaw Library of the Medical College of Virginia, Richmond, are listed below. These are available to our readers under usual library rules.

Ackerman—Surgical pathology. 1953.
Advances in pediatrics. Vol. 6, 1953.
Cahoon—Formulating x-ray technics. 3d ed., 1953.
Chamberlain—Symptoms and signs in clinical medicine. 5th ed., 1952.
Eccles—The neurophysiological basis of mind. 1953.
Ford—Diseases of the nervous system in infancy, childhood and adolescence. 3rd ed., 1952.
Grulee & Eley—The child in health and disease. 2d ed., 1952.
Holt & McIntosh—Holt pediatrics. 12th ed., 1953.
Horder & Gow—The essentials of medical diagnosis. 2d ed., 1953.
Josiah Macy, Jr. Foundation—Adrenal cortex. 1953.
Lindskog & Liebow—Thoracic surgery and related pathology. 1953.
Mallett—Handbook of anatomy & physiology for x-ray technicians. 1952.
Marple & Wright—Thromboembolic conditions and their treatment with anticoagulants. 1950.
Quick—Physiology and pathology of hemostasis. 1951.
Recent progress in hormone research. Vol. 8, 1953.
Selle—Body temperature: its changes with environment, disease and therapy. 1952.

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THE MEDICAL SOCIETY OF VIRGINIA

COMMITTEE REPORTS RECEIVED TOO LATE TO APPEAR IN THE SEPTEMBER MONTHLY

Rehabilitation

The Committee of Rehabilitation, also acting as Professional Advisory Committee for the Vocational Rehabilitation Service, State Department of Education, has continued its several functions during the past year.

Individual members, located in various geographical areas of the state and representing various specialty fields, have served as consultants to the staff of the Rehabilitation Agency throughout the year. Professional advice was given on individual cases involving medical problems. In addition professional guidance was provided on the revision of and additions to the professional fee schedule.

The Committee encouraged the expansion of the Physical Evaluation Program. The several Units of the Program is to determine more effectively the rehabilitation prospects of severely disabled cases and those with multiple medical problems through the team approach technique. In addition to the Norfolk Unit, others have been established in Newport News, Richmond, Roanoke, and at Woodrow Wilson Rehabilitation Center, Fishersville. Each of these Units is meeting periodically as the need arises. The results reported during the past year clearly demonstrate the advantages of this approach to case appraisal and further expansion of the Physical Evaluation Program is anticipated.

A regular meeting of the Rehabilitation Committee was held at the John Marshall Hotel on March 22. In addition to regular committee members, others present were: R. N. Anderson, Director, Vocational Rehabilitation and Special Education; Dr. J. M. Mennell and Mr. F. O. Birdsall of Woodrow Wilson Rehabilitation Center; Dr. Herbert W. Park, Physiatrist, Department of Physical Medicine, Medical College of Virginia Hospital; Messrs. Corbett Reedy and Floyd H. Armstrong of the State Rehabilitation Service.

Encouraging reports were submitted on the accomplishments of the Woodrow Wilson Rehabilitation Center and the effectiveness of the several Units of the Physical Evaluation Program.

Mr. Robert I. Howard, Executive Secretary-Treasurer and Director of Public Relations, attended a portion of the meeting and informed the Committee of tentative plans for a session on "Rehabilitation" at the next annual meeting of the Virginia Medical Society. The Committee expressed its hearty approval of the plan.

Respectfully submitted,

ROY M. HOOVER, M.D., *Chairman*
GEORGE A. DUNCAN, M.D.
J. R. BLALOCK, M.D.
LEROY SMITH, M.D.
FRANK B. STAFFORD, M.D.
W. E. DICKERSON, M.D.
G. S. FITZ-HUGH, M.D.
FLETCHER J. WRIGHT, M.D.
A. L. CARSON, JR., M.D.
RENO PORTER, M.D.
EDWARD E. HADDOCK, M.D.
CHARLES L. SAVAGE, M.D.

To Confer With The State Board of Nurse Examiners

The committee to confer with the State Board of Nurse Examiners appointed by Dr. James L. Hamner, President of the Medical Society of Virginia, October, 1952, at the time of this writing, has had no formal meetings and has had no request, either from physicians or nurses, to consult.

Parenthetically, the present trends in nursing education and nursing practice being what they are, one might fairly raise the question whether the Committee should (1) try to do something about it or (2) acquiesce silently into the oblivion of futility.

Not being disposed to initiate a crusade, the Committee has no further report to submit at this time.

Signed,

R. V. BUXTON, M.D.
J. A. SHACKELFORD, M.D.
E. L. LOWENBERG, M.D.
F. JOHNS, M.D.
M. H. HARRIS, M.D.
J. M. HABEL, M.D.
C. B. MORTON, M.D., *Chairman*

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Medical Society of Virginia Cancer Committee

Chairman, George Cooper, Jr., M. D.

Albemarle Hotel Building, Charlottesville, Va.

Reprints of this and preceding Bulletins may be obtained from this office

October 1, 1953

Physical Diagnosis

In cancer diagnosis, as in all medical diagnosis, accuracy is in direct proportion to the completeness of the physical examination. It is difficult for the physician who has a large, busy practice to carry out routinely as complete an examination as he would like. And in this day of free use of and great reliance upon laboratory diagnosis, the physician is more apt than ever before to neglect to exhaust the possibilities of physical diagnosis. The inevitable result is avoidable inaccuracy.

Case I. A middle-aged woman developed a mucopurulent left-sided nasal discharge. At times, the material was bloody. Her physician examined her nose and throat but did not use a mirror to visualize the nasopharynx. Finding no explanation, he decided she must have sinusitis. Roentgen examination of the sinuses supported the diagnosis by revealing abnormal density in the left ethmoid capsule and some thickening of the mucous lining of the left antrum. Antibiotics were given from time to time for six months. There was transient decrease in the discharge, but on the whole, it grew more profuse, foul, bilateral, bloodier, the throat grew dry, and there was gradual impairment of hearing in the left ear. An episode of epistaxis lead to a consultation with a second physician. He completed the physical examination by doing a mirror inspection of the nasopharynx. A bulky, left-sided tumor was only too evident. The biopsy report was undifferentiated carcinoma.

Case II. An elderly woman developed pain in the left side of the base of the tongue. She consulted her physician, who inspected the tongue but did not palpate it. There was nothing on the tongue, but he did find foul, caseous material extruding from the tonsillar crypts. He ascribed her pain to tonsillitis. At his suggestion, a second physician performed a tonsillectomy. The latter also neglected to palpate the painful tongue. In spite of the tonsillectomy, the pain grew steadily worse. A third physician was called in. He palpated the painful area and promptly located a hard tender submucosal mass. Biopsy revealed an undifferentiated carcinoma. Incidentally submucosal carcinoma in the base of the tongue is one of the few cancers sporting enough to produce pain as an initial and early symptom.

Case III. A woman in the menopausal age was given estrogens for intermenstrual spotting. No pelvic examination was done. When the spotting grew worse, a second physician was consulted. He did a bimanual pelvic examination and found an enlarged, nodular corpus. Satisfied, he omitted a speculum examination of the cervix, and performed a supravaginal hysterectomy, partially removing a fibroid uterus. He was shocked when the pathologist informed him he had cut through an epidermoid carcinoma. He then performed the speculum examination and found a small cervical ulceration. Biopsy showed the same neoplastic cells that were in the operative specimen.

Case IV. A middle-aged man developed painful, bleeding hemorrhoids. He consulted a physician who limited his investigation to inspection of the anus, and then performed a hemorrhoidectomy. Bleeding persisted. Several months later, a second physician was consulted. He performed a digital rectal examination, and at once discovered a stony hard, nodular tumor several inches above the sphincter. Biopsy revealed an adenocarcinoma.

Despite tremendous advances in diagnostic methods and techniques, there is still no substitute for a thoughtful, complete investigation of a patient's complaints by physical diagnosis.

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EDITORIAL

Howard Taylor Ricketts (1871-1910) and Rickettsiae

THE rickettsiae and viruses have taken their toll of martyrs of science, and their study has opened a new era in medicine. These infectious agents are obligate parasites so small that it is difficult to see them with ordinary microscopes and many are so small that they readily pass through earthenware filters. As demonstrated by special microscopes they vary greatly in size and shape. It is difficult to obtain enough material in pure form to study them chemically but one, at least, the tobacco-mosaic virus, appears to be nothing more than a crystal of nucleoprotein. Outside of their host cells they exhibit no sign of life—respiration, metabolism, nor reproduction—yet when united to their specific host cells they cause some of the most dread diseases of man and beast. Among them may be mentioned: Viral encephalitides (St. Louis, Japanese, Australian, western equine, eastern equine, etc.), rabies, poliomyelitis, infectious hepatitis, serum hepatitis, common cold, atypical pneumonia, influenza, smallpox and

Howard Taylor Ricketts, M.D.
Courtesy of the Armed Forces Medical Library

vaccinia, psittacosis, trachoma, measles, rubella, epidemic keratoconjunctivitis, herpes zoster, mumps, infectious mononucleosis, pretibial fever, Colorado tick fever, yellow fever, Rift Valley fever, dengue, typhus fever (louse-borne), typhus fever (flea-borne), Brill's disease, Rocky Mountain spotted fever, etc., scrub typhus, Q fever, trench fever, and foot-and-mouth disease (in cattle).

Many of these diseases confer lasting immunity, others an immunity of shorter duration and others no immunity at all. As a group they afford an opportunity for worthwhile studies, but unfortunately, adequate study of these diseases requires elaborate

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techniques and highly trained personnel that are not available in ordinary clinical laboratories. One must have a range of laboratory animals from mice to monkeys, and thousands of chick embryos. One might well call this era the monkey-chick-embryo-era. Even to make a diagnosis, such equipment is needed. To decide whether the disease that Brill described in New York were real typhus or not, epidemiologists with their laboratory animals inoculated with their particular strains of disease from New York and the Rocky Mountains met in Mexico City where typhus was endemic. It was such a conclave of men, monkeys, and infected arthropods that cost Ricketts his life.

Typhus fever has always been a dangerous disease for doctors and nurses. In some epidemics it has killed literally hundreds of them. In my student days there was a small outbreak—six cases, I believe—in Baltimore. They were carefully guarded in the isolation ward and the students were not allowed even to see them. Of the six men working with Ricketts in Mexico, three contracted the disease, two of whom, Ricketts and Conneff, died. The causative agent, *Rickettsia prowazekii* was named for Ricketts and S. von Prowazek, an Austrian, who also lost his life while studying the disease.

Howard Taylor Ricketts was born on an Ohio farm. Early in his life, his parents moved to Champaign County, Illinois. At sixteen he entered the Academy of Northwestern University and at nineteen the University proper. In his sophomore year he decided to study medicine. It is said that a fellow student, who afterwards became his wife, had much to do with the decision. His family now moved to Lincoln, Nebraska, and in 1894 young Ricketts took his Bachelor's degree at the University of Nebraska. He graduated in medicine from Northwestern in 1897 and served as interne at Cook County Hospital until 1899 when he became a fellow in cutaneous pathology in Rush Medical College.

In 1900, Ricketts married Myra Tubbs who supported him in his work with rare devotion, keen interest, and steady encouragement, and bore him two children, a son and a daughter.

Returning in 1902 from a year's visit to European laboratories, he came to the University of Chicago as instructor in the newly founded Department of Pathology and Bacteriology. Later he became assistant professor and, in the early part of 1910, he accepted the chair of pathology at the University of Pennsylvania, a position he did not live to fill.

His earlier researches, begun in 1902 on blastomycosis and immunological problems, are marked by thoroughness and directness and by clear and forceful reasoning. He took up the study of Rocky Mountain spotted fever in the spring of 1906 as a pastime during an enforced holiday on account of overwork. The peculiarities of the disease intrigued him. It is sharply limited geographically and occurs only in the spring. For some time it was vaguely thought to be related in some way to the bite of a tick. Ricketts not only found the particular tick involved but demonstrated the causative agent in the patient, and in the body, eggs, and offspring of the infected tick. The hereditary transmission of spotted fever virus in the tick was an entirely new concept.

His work on Rocky Mountain spotted fever naturally led to typhus fever as their problems are similar. In December 1909 he went to Mexico to study typhus fever. At the time the cause of the disease had not been discovered. Nicolle (1909) in Tunis was able to transmit the disease to the chimpanzee by injecting the blood of human patients, but was able to infect the macacus monkey only by using the body louse. In Mexico, Anderson and Goldberger (1909) had no difficulty in infecting a macacus and a capuchin

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by direct inoculation with virulent human blood. This was apparently laboratory confirmation of the widespread belief that the old world typhus and the new world typhus were two different diseases. Shortly after he demonstrated the actual cause, Ricketts contracted the disease and on May 3, 1910, he died in a tent on the grounds of the American Hospital. President Diaz of Mexico directed that the professors and students of the National Medical School be present on the occasion of the removal of the body to Chicago, that the medical school and institute of bacteriology be draped in mourning for three days, and that the laboratory in which Professor Ricketts had made his investigations be named "The Howard T. Ricketts Laboratory". The Chicago Pathological Society published as a memorial volume "Contributions to Medical Science by Howard Taylor Ricketts 1870-1910".

Ricketts is described by Professor Hektoen, his chief at Chicago, as a modest, unassuming man of great determination and of the highest character, loyal and generous, earnest and genuine in all his doings—a personality of unusual and winning charm.

SOCIETIES

Virginia State Board of Medical Examiners. Following is a list of those licensed by the Board at its examinations in Richmond, June 17-20, 1953:

LICENSED TO PRACTICE MEDICINE AND SURGERY BY EXAMINATION

Dr. Simeon Huey Adams, Gastonia, N. C.
Dr. Betty Austin Agee, Warrenton
Dr. Richard Bryan Agee, Warrenton
Dr. Pedro Juan Alfaro, Caparra Heights, Puerto Rico
Dr. William Glenn Allen, Jr., Charlottesville
Dr. Robert Francis Barbe, Abingdon
Dr. Cavitt Kinkle Bartley, Charlottesville
Dr. James Gordon Beach, Jr., Gary, W. Va.
Dr. Eugene Bene, Staunton
Dr. Wesley Clifford Bernhart, Springfield, Ohio
Dr. Charles Lee Boatwright, Jonesville
Dr. Lester Ray Bond, Roanoke
Dr. Herman Wallace Brubaker, Roanoke
Dr. William Harvey Cabaniss, Jr., Athens, Ga.
Dr. Carmen Margarita Caceres, San Juan, Puerto Rico
Dr. James Monroe Carter, McKeesport, Pa.
Dr. George Edward Chappell, Richmond
Dr. Walter Scott Chisholm, Jr., Denver, Colo.
Dr. Kenneth Melvin Clements, Richmond
Dr. Alvin Cohen, Petersburg
Dr. William Patrick Coleman, Charlottesville
Dr. Alvin Eugene Conner, Manassas
Dr. Catherine Elizabeth Craun, Richmond
Dr. Pleasant Paul Deaton, San Antonio, Texas
Dr. Sterling Randolph Decker, Charlottesville
Dr. Robert Bailey Deitchman, Martinsville
Dr. Patrick Campbell Devine, Charlottesville
Dr. Ronald Burton Dietrick, Richmond
Dr. Mary Catherine Duckgeischel, Santa Clara, Cal.
Dr. Robert Kent Dyer, Roanoke
Dr. Walker Marshall East, Clifton Forge
Dr. Julia Emmett Edmunds, Charlottesville
Dr. Ernest Gray Edwards, Hampton
Dr. Cecil Franklin Evans, Jr., Richmond
Dr. Channing Lester Ewing, Pensacola, Florida
Dr. Hugh Lanier Farrior, Concord, N. C.
Dr. Morris Fine, Salt Lake City, Utah
Dr. William Latane Flanagan, Naval Base, S. C.
Dr. William Paul Fletcher, Portsmouth
Dr. James Stephenson Foster, Dante
Dr. Earl Russell Fox, Naruna
Dr. John Happer Furr, Washington, D. C.
Dr. James Harry Gamble, Charlottesville
Dr. Robert Daniel Gardner, Flemington, N. J.
Dr. Calvin Sampson Garrett, Norfolk
Dr. William Neville Gee, Jr., Norfolk
Dr. Frederick True Given, Jr., Chase City
Dr. Harvey Winfree Goode, Jr., Powhatan
Dr. Paul Wiley Gordon, Jr., Clifton
Dr. Julius Griffin, Los Angeles, Cal.
Dr. Douglas Meriwether Griggs, Jr., Charlottesville
Dr. Robert Burwell Groves, Jr., Galveston, Texas
Dr. Warren Cleaton Hagood, Norfolk
Dr. David Goodsell Hall, III, Charlottesville
Dr. Tibor Ham, Columbia, S. C.
Dr. Euclid M. Hanbury, Jr., Montreal, Canada
Dr. Edward Harrison Hancock, Jr., Charlottesville
Dr. Arthur Garner Harden, Virginia Beach
Dr. Charles Pinchbeck Harwood, Richmond
Dr. John Thomas Hearn, Jr., Fredericksburg
Dr. Ernest Conrad Hermann, Richmond
Dr. Jesse Robinson Hicks, Charlottesville
Dr. Royce Linton Holsey, Norfolk
Dr. Raymond Cottrell Hooker, Jr., Richmond
Dr. John Shelton Horsley, III, Richmond
Dr. Edward Claiborne Irby, Blackstone
Dr. Harry Isaac Johnson, Jr., Salem
Dr. John Walter Carlysle Johnson, Jr., Charlottesville
Dr. William Raymond Johnson, Jr., Mount Sidney
Dr. Robert Henry Jordan, Norfolk

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Dr. Louis Eugene Jorel, Phoenixville, Pa.
Dr. Michael John Keith, Williamsburg
Dr. Verbon Eric Kemp, Jr., Richmond
Dr. Carl Peter Kremer, Jr., Rochester, N. Y.
Dr. David Benson Kruger, Charlottesville
Dr. Walter Fitch Manley, Jr., Roanoke
Dr. Moir Glenwood Martin, Hillsville
Dr. Philip Royden Mason, Richmond
Dr. George Bernard Massie, Pounding Mill
Dr. Arch Thomas McCoy, Richmond
Dr. Leroy Sutter McDaniel, Richmond
Dr. Thomas David McDonald, Roanoke
Dr. Wilmore Randolph McLeod, Glen Allen
Dr. Harvey Edward Melton, Springfield, Ohio
Dr. Fred Neal Mitchell, Charlottesville
Dr. Daniel Nathan Mohler, Jr., Charleston, W. Va.
Dr. Charles Hunter Montgomery, Charlottesville
Dr. Samuel Stuart Morrison, Springfield, Ohio
Dr. George Edward Mowry, Wicomico
Dr. John Girardwau Murray, Jr., Richmond
Dr. John Jennett Neal, Jr., Danville
Dr. James Linwood Odom, Norfolk
Dr. John A. Opal, Clintwood
Dr. Sheff Daniel Orlinger, Jr., R.F.D. 1, Coeburn
Dr. Ross Moore Orr, Jr., Columbus, Ohio
Dr. Patsy Ruth Phelps, Baltimore, Md.
Dr. Frank Wilson Price, Charlottesville
Dr. Jean Fensom Ragsdale, Richmond
Dr. Norman Clydlee Ratliffe, Richmond
Dr. John Rebman, III, Richmond
Dr. Arthur McKinley Reynolds, Jr., Portsmouth
Dr. Charles Wellington Richardson, St. Petersburg, Fla.
Dr. Malcolm Kauffman Rosenbaum, Washington, Pa.
Dr. Lawrence Charles Rosenberg, New York, N. Y.
Dr. Malcolm Lee Rosenblatt, Boston, Mass.
Dr. Frank Edward Rowell, Norfolk
Dr. George Straughan Schools, Richmond
Dr. William Allen Scoggin, Charlottesville
Dr. Pierre B. Scott, Seattle, Washington
Dr. George Oscar Shipp, Norfolk
Dr. John Calvin Shipper, Martinsburg, W. Va.
Dr. Bobby Long Smith, Raleigh, N. C.
Dr. James Roger Smith, Ashland
Dr. Richard Holloday Smith, Jr., Nashville, Tenn.
Dr. William Kyle Smith, Jr., Annapolis, Md.
Dr. Louis Max Sod, Norfolk
Dr. Alger Rixey Southall, Jr., Pamplin
Dr. Henry Sumpter Spencer, Richmond
Dr. Philip Martin Sprinkle, Seattle, Washington
Dr. Walter Jackson Stanford, St. Petersburg, Florida
Dr. Thomas Peirson Stratford, Richmond
Dr. Luther Marcus Talbert, Charlottesville
Dr. Paul Alexander Tanner, Jr., Quitman, Ga.
Dr. Terry Finley Tanner, Quitman, Ga.
Dr. William Burwell Tarry, Jr., Brookneal
Dr. Raymond Jay Thabet, Charleston, W. Va.
Dr. Charles Gerhard Thedieck, Jr., Suffolk
Dr. John Vernon Torbert, Jr., Chesapeake
Dr. Charles James Townsend, Springfield, Ohio
Dr. Robert William Trever, Arlington
Dr. Frederik Van Den Branden, Williamsburg
Dr. John Herndon Vansant, Charlottesville
Dr. Emery Mittchell Ward, Charlottesville
Dr. Robert Edward Ware, Worcester, Mass.
Dr. John William Watson, Philadelphia, Pa.
Dr. William Greene Way, Philadelphia, Pa.
Dr. Thomas Harry Webb, Portsmouth
Dr. James Hugh Wiley, Richmond
Dr. George Lee Wilkinson, South Boston
Dr. Samuel Nimrod Whitacre, Cincinnati, Ohio
Dr. Walter Raleigh Whitehurst, Jr., Baltimore, Md.
Dr. Woodrow Wilson Wren, Springfield, Ohio
Dr. Allan William Wright, Richmond

HOMEOPATHIC MEDICINE AND SURGERY

Dr. Edmundo Morales, Washington, D. C.

LICENSED TO PRACTICE MEDICINE AND SURGERY BY RECIPROCITY

Dr. Donald Carl Anderson, Pensacola, Fla.
Dr. Robert H. Armstrong, Annapolis, Md.
Dr. Thomas Marshall Arrington, Richmond
Dr. Herbert Mead Bowlby, Staunton
Dr. Bernard Bressler, Alexandria
Dr. Henry Thomas Brobst, Roanoke
Dr. Raymond Nathan Brown, Washington, D. C.
Dr. Robert Junius Brown, III, Winston-Salem, N. C.
Dr. Frank Brian Clare, Portsmouth
Dr. Moses Cohen, Queens County, N. Y.
Dr. George Henry Cook, Chincoteague
Dr. William Hurlbert Cooper, Washington, D. C.
Dr. Armando Ralph Coppola, Newport News
Dr. Francis Willson Daily, Roanoke
Dr. Charles R. Derrickson, Arlington
Dr. Thomas Isom Durm, Portsmouth
Dr. Norman E. Dyhrberg, Roanoke
Dr. Louis Zephirin Fauteux, Jr., Falls Church
Dr. Gerald J. Fisher, Arlington
Dr. Marion White Fisher, Sandston
Dr. Ann Conway Fred, Fort Lee
Dr. Lawrence Taylor Giles, Warrenton
Dr. Frederick Richard Gilmore, Durham, N. C.
Dr. Samuel J. Hall, Boston, Mass.
Dr. Brooks Lynn Hargrove, Jr., Portsmouth
Dr. Roy Belmont Hargrove, Jr., Farmville
Dr. Eugene Witter Higgins, Roanoke
Dr. Isham Earl Holmes, Staunton
Dr. Fritz Milton G. Holmstrom, Arlington
Dr. Wayne Stanford Hume, Fort Monroe
Dr. Charles Jared Ingersoll, Dahlgren
Dr. Charles Southward Ireland, Norfolk
Dr. Thomas Nelson Page Johns, Richmond
Dr. David Strand Johnsen, Washington, D. C.
Dr. Arthur Sanford Kaplan, Norfolk
Dr. Richard Eugene DeMonbrun Kepler, Honolulu, Hawaii
Dr. Michael F. Lapadula, Arlington
Dr. James John Lawson, Jr., Bayonne, N. J.
Dr. Carroll Hoyt Lippard, Lynchburg

Dr. Kurtland Herrick Lord, Richmond
Dr. Thomas Edward Manly, Paterson, N. J.
Dr. Walter Sidney Matthews, Jr., Falls Church
Dr. Charles Francis McCaffrey, Arlington
Dr. Kenneth Earl McIntyre, Marion
Dr. Willys Moore Monroe, Norfolk
Dr. Paul William Morgan, Waynesboro
Dr. Kenneth Newton Payne, Hampton
Dr. Irwin Elwood Phillips, Bristol, Tenn.
Dr. Douglas Theodore Prehn, Arlington
Dr. George Sylvester Price, Jr., Martinsville
Dr. Charles Danny Pruett, Bluefield, W. Va.
Dr. F. Turner Reuter, Washington, D. C.
Dr. William Davis Richards, Richmond
Dr. Henry Moore Rogers, Jr., Wilson, N. C.
Dr. Sidney Rosenbaum, Arlington
Dr. Arnold M. Salzberg, Richmond
Dr. Walter Wesley Sawyer, Jr., Norfolk
Dr. Howell H. Sherrod, Johnson City, Tenn.
Dr. Richard Harrison Small, Falls Church
Dr. James Joseph Thorpe, Falls Church
Dr. Walter Weddle Vaughan, Durham, N. C.
Dr. Earl Edward Walker, Jr., Richmond
Dr. Flotilla Watkins, Roanoke
Dr. Charles Richard Webb, Washington, D. C.
Dr. Louise Oftedal Wensel, Washington, D. C.
Dr. W. Leonard Weyl, Arlington
Dr. Gordon A. Zimmerman, Arlington

Eight others were licensed to practice osteopathy, chiropody, and chiropractic.

Medical Society of Northern Virginia.

At the regular meeting of this Society on August 11th, there were present twenty-five members and their wives. After a pleasant dinner meeting, the professional session was held and consisted of a lecture and a motion picture on the Medical Aspects of Civilian Defense, the speaker being Dr. W. R. Southward of the State Department of Health, Richmond. At a business session, five doctors were admitted to membership.

Dr. Elizabeth Sherman of Front Royal is president and Dr. William C. Humphries of Woodstock is secretary-treasurer.

Fairfax County Medical Society.

At a meeting of this Society held on September 8, the following officers were elected for the coming year: President, Dr. Claude Cooper, Annandale; vice-president, Dr. Paul Kemp, Falls Church; secretary, Dr. Robert Hunt, Falls Church; treasurer, Dr. Robert Rounds, Falls Church.

A committee has been appointed to make plans for the annual dinner dance to be held next month.

ALICE H. KIESSLING, M.D.,
Publicity Chairman.

NEWS

The Medical Society of Virginia

Is to hold its 106th annual meeting at Hotel Roanoke, Roanoke, October 18-21, under the presidency of Dr. James L. Hamner of Mannboro. The programs which are being mailed all members show that many papers on timely subjects are to be discussed. Round tables and panel discussions will be interesting features. On Tuesday evening will be the social hour, and banquet with entertainment. Arrangements have been provided for the pleasure of the ladies during and after the Auxiliary meetings. Excellent accommodations are to be had in Roanoke, and the meeting there promises to be a banner one as usual.

Southern Medical Association.

Coming within a week following the meeting of The Medical Society of Virginia will be the Southern Medical meeting in Atlanta, Georgia, October 26-29. This is always enjoyed by Southern doctors and their wives. The Municipal Auditorium will be general headquarters and is conveniently located within walking distance of all downtown hotels. Dr. Walter C. Jones of Miami, Florida, is this year's president. If rooms have not been reserved, get in touch at once with the Housing Bureau of the S.M.A., 801 Rhodes-Haverty Building, Atlanta 3, Georgia, for information, stating the type and price of accommodations desired.

News from University of Virginia School of Medicine.

The fall series of Monday Evening Medical Lectures will open October 5, with the following speakers: Dr. Louis G. Welt, Dr. William H. Sebrell, Jr., Dr. Carl V. Moore and Dr. Loyal Davis. The November speakers will likewise include a number of prominent out-of-State speakers.

Eight new members have been appointed to the University of Virginia School of Medicine faculty, Dean Thomas H. Hunter announced recently.

Dr. Oscar A. Thorup, Jr., a medical graduate of the University in 1946 and former chief resident in

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internal medicine who has been serving during the past year as research fellow in internal medicine at the University of North Carolina, has been appointed Assistant to the Dean and Instructor in Internal Medicine.

New assistant professors include Dr. Julian R. Beckwith, a medical graduate of the University in 1936 and former chief of the Medical Service of the C. & O. Hospital in Clifton Forge, Va., to the Department of Internal Medicine; Dr. W. A. H. Councill, Jr., a medical graduate of the University in 1947, to the Department of Urology; and Dr. Eugene D. Brand, formerly of the University of Utah, to the Department of Pharmacology.

Newly appointed instructors are Dr. Warren Gregory, a medical graduate of the University in 1950, to the Department of Pediatrics; Dr. A. Alan Humphries, a graduate of Emory and Princeton universities, to the Department of Anatomy; and Dr. Leroy S. Pearce, a graduate of Vanderbilt University, to the Department of Pathology.

Dr. Francis H. McGovern, a graduate of the University of Pennsylvania who is engaged in private practice in Danville, Va., has been appointed Clinical Assistant Professor of Otolaryngology.

De Paul Hospital Expresses Appreciation of Dr. Martin.

De Paul Hospital, Norfolk, at a meeting in July 1953, adopted the following resolution honoring Dr. Walter B. Martin:

We wish to present a resolution to do honor to a member of our staff who has achieved great distinction,

WHEREAS, Dr. Walter B. Martin has been made President-Elect of the American Medical Association and has brought tremendous honor to himself, the Staff of De Paul Hospital, the City of Norfolk, and the State of Virginia, be it

RESOLVED, that the Staff of De Paul Hospital, wishing to acknowledge this honor, has prepared this Resolution and recommends that this Resolution be spread upon the minutes of the Staff of De Paul Hospital and a copy be sent to Dr. Walter B. Martin and to the Virginia Medical Monthly.

GEORGE W. SCHENCK, M.D., *Chairman*
JOHN W. OAST, M.D.

Credits to Be Given Air Force Reserve Officers.

Medical Service Officers of the Air Force Reserve who attend the Association of Military Surgeon's Annual Convention in Washington, D. C., November 9-11, 1953, will receive Reserve training credits consisting of one (1) point for each day's attendance. A representative of the Surgeon General's Office, Headquarters, USAF, will be present during the convention to certify attendance for each Medical Officer, Dental Officer, or Nurse claiming these training points. Officers must participate for a minimum of two hours each day. The maximum amount of credit that can be earned is three points.

"Air Force Reserve point credits are not normally awarded for attendance at associations, conventions, and sessions, but it is believed (by Headquarters) that this specific convention warrants points since the topics, lectures and exhibits will be of material training value to Medical Service Officers attending."

The Gill Memorial Hospital Spring Congress

Will be held in Roanoke, Virginia from April 5 through April 10, 1954. Among the guest speakers will be:

Dr. John Foster	Leeds 1, England
Dr. Edwin B. Dunphy	Boston, Mass.
Dr. Charles L. Schepens	Boston, Mass.
Dr. Harvey E. Thorpe	Pittsburgh, Pa.
Dr. Hayes Martin	New York, New York
Dr. Louis Clerf	Philadelphia, Pa.
Dr. Alson E. Braley	Iowa City, Iowa
Dr. Hyman S. Sugar	Detroit, Michigan
Dr. George Merriam, Jr.	New York, New York
Dr. Victor Goodhill	Los Angeles, Calif.
Dr. John McLean	New York, New York
Dr. John R. Richardson	Boston, Mass.
Dr. M. Elliott Randolph	Baltimore, Maryland
Dr. Edmund B. Spaeth	Philadelphia, Pa.
Dr. Winchell Craig	Rochester, Minnesota
Dr. George Murgatroyd	Baltimore, Maryland
Dr. Donald Proctor	Baltimore, Maryland

Southern Electroencephalographic Society.

The next annual meeting of this Society will be held in Atlanta, Georgia, October 28-29, 1953, at the Ansley Hotel. The Society will also meet jointly with the section of Neurology and Psychiatry of the Southern Medical Association.

Dr. Isadore Samuel Zfass, psychiatrist and neurologist of Richmond, was elected president of the Southern Electroencephalographic Society at its annual meeting held at Miami Beach, Florida. Dr. Zfass has been one of the pioneer electroencephalographers in the South, and is the only physician in Virginia who is certified by the American Board of Qualifications in Electroencephalography.

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The Section on Clinical Cardiology

Of the American Heart Association will sponsor a two-day scientific program at the Conrad Hilton Hotel in Chicago on April 3 and 4, 1954. This program will constitute a portion of the Annual Meeting of the American Heart Association and immediately precedes the Annual Sessions of the American College of Physicians. The meeting will be open to all members of the medical profession. Dr. Wright R. Adams of Chicago is Chairman of the Program Committee. Members of the American Heart Association who wish to present papers should send a 250-300 word abstract of the proposed paper to Dr. Charles D. Marple, Medical Director, American Heart Association, Inc., 44 East 23rd Street, New York 10, New York. *All papers should be on subjects of distinct clinical interest. The deadline for the receipt of abstracts is January 1, 1954.*

Bought Any New Medical Books Lately?

Your older volumes would be gratefully received by the physicians, hospitals, and universities of Israel. Especially needed are books in all medical specialties.

If you can spare any of these books which are in good condition and published since 1940, please send them by prepaid parcel post to: Books for Israel, 115 King Street, New York 1, N. Y.

NOTE: Up to 70 lbs. may be sent by parcel post, at 8¢ for the first pound, and 4¢ each additional pound, marked "BOOK RATE." Please give return address of individual or organization, so that your gift may be acknowledged. Funds for shipment from New York to Israel have been provided under Point IV, U. S. State Department, sponsors of this project.

Dr. I. Floyd Nesbitt,

Obstetrician formerly of Newport News, has moved to Durham, North Carolina.

Dr. Thomas S. Ely,

Of Jonesville, was elected the new commander of the Virginia American Legion, at is meeting in Roanoke, the middle of August.

Dr. Carl I. Pirkle

Has been transferred from Lewisburg, Pennsylvania, to the U. S. Penitentiary, Atlanta, Georgia, as chief medical officer for that institution, effective September 1. Dr. Pirkle was for a number of years with a similar institution in Prince George, Virginia.

New Manager at McGuire VA Hospital, Richmond.

Dr. Glen W. Doolen, who has been connected with the Tuberculosis Division of the VA's central office in Washington, D. C., has been named manager of the Veterans Administration Hospital in Richmond, and entered upon his new duties the 1st of September. He took over the position formerly held by Dr. John G. Hood, who was transferred to become manager of the Bronx Veterans Administration Hospital in New York City.

Pan-American Academy of General Practice.

Through the courtesy of the Government of Colombia, expressly, the University of Antioquia (celebrating their 150th year of its foundation) and the Dean of the Medical School, the Pan-American Academy of General Practice, has been invited to hold its annual Congress in the city of Medellin, Colombia, S.A., February 15th to 20th, 1954. Following Medellin, there will be a visit to Bogota, Barranquilla and Montega Bay, Jamaica, the complete trip being from February 15th to the 28th. The Congress activities will be divided into sections, representing the various fields of Medicine, Surgery and Pharmacy.

Those participating in this trip will be declared Guests of Honor, and there will be extensive festivities for their benefit. It also offers an opportunity for a delightful holiday in glamorous, exotic and historic Colombia.

Transportation at a special all inclusive rate, via, plane or steamship, will be available to those participating. Registration must be made through Dr. Arturo Martinez, Secretary, 500 West End Avenue, New York, N. Y.

American College of Surgeons.

For the first time since 1914, when a Clinical Congress was held in London, England, that city will be host to a Sectional meeting of the American College on May 17 to 19, 1954. It is anticipated that more than 500 Surgeons from Canada and the United States will attend. The Royal College of Surgeons plans not only an imposing series of lectures but also some receptions to which the visiting surgeons are cordially invited.

Clinical Seminar.

The Bluefield Sanitarium, Bluefield, West Virginia, is holding its first annual clinical seminar in

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the staff room of the Sanitarium, October 2, at 2:00 p.m. A varied program will be presented by the staff members. Dr. George T. Harrell, Jr., Professor of Medicine at the Bowman-Gray Medical School, Winston-Salem, North Carolina, will be the guest speaker at the banquet to be held that evening at the Bluefield Country Club. His topic will be "Myxedema".

Trudeau Sanatorium,

Trudeau, New York, announces that it will mail a pamphlet to physicians east of the Mississippi interested in diseases of the chest, to bring them up-to-date on the Sanatorium, which celebrates his seventieth anniversary next year. Copies of the pamphlet are available by writing Dr. Roger S. Mitchell, Clinical Director, Trudeau Sanatorium, Trudeau, N. Y.

Wanted:

Physician for the Staff of the DeJarnette State Sanatorium, Staunton, Virginia. This is a psychiatric hospital of approximately 180-bed capacity. Starting salary $8,400.00 to $8,784.00. Apply to the Superintendent. (Adv.)

For Sale.

Retiring physician's home and equipped rented office in Cape Charles, Virginia. Address P. O. Box 304, that town (*Adv.*)

For Rent—

Four-room medical suite, excellent location, with waiting room, fluoroscope, laboratory and physiotherapy to share. Parking space and all utilities provided. Also complete 2 chair dental suite.

Apply to R. G. McAllister, M. D., 1016 W. Franklin Street, Richmond 20, Va. (*Adv.*)

Wanted:

1. A physician for contract practice in coal camp;
2. A physician for private hospital, preferably internal medicine.

Address Dr. W. C. Elliott, Lebanon General Hospital, Lebanon, Virginia. (*Adv.*)

OBITUARIES

Dr. Oscar Raymond Yates,

Suffolk, died July 15 in Obici Hospital, Suffolk, after a six weeks illness from coronary occlusion and myocardial failure. He graduated from the Medical College of Virginia in 1916, and had practiced in Suffolk since, with the exception of military service during World War I.

He was very active in civic, fraternal, and church activities, and among other recognitions for his community services, he was the recipient of the Silver Beaver award for outstanding work with the Boy Scouts, and was given a special citation by the Lions Club for performing without charge a large number of tonsillectomies for underpriviledged children. At the time of his death he was president of the Obici Hospital medical staff. He is survived by his wife, a daughter and a son.

Dr. Robert Dennis Caldwell,

Widely known pathologist of Lynchburg, died August 18, at his home, after an extended illness. He was 57 years of age and graduated from the Medical College of Virginia in 1919. He served at hospitals in Richmond for a year before locating in Lynchburg where he had since made his home. His wife and several brothers and sisters survive him. Dr. Caldwell was a member of his local, State and several other societies, including the American Medical Association. Burial was in Lewisburg, W. Va.

Dr. William F. Mercer,

Richmond, a graduate of the Medical College of Virginia in 1882, and said to be its oldest graduate, died at his home in this city, September 4, at the age of 91. He had been in bad health for sometime and had retired from practice some years ago. He was a prominent ophthalmologist for many years and was formerly a member of many societies connected with his specialty. A daughter survives him.

Dr. Alexander G. Brown, Jr.

As we go to press, we learn with sorrow of the death of Dr. Brown who, as chairman of the Publication Committee of The Medical Society of Virginia, became editor of the VIRGINIA MEDICAL MONTHLY in April 1919 and served in this office most efficiently until October 1933 when he asked to be relieved of this position because of pressure of other duties.

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